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PRE CLINICAL AND CLINICAL STUDY ON

MATHUMEGAM

(DISSERTATION SUBJECT)

For the partial fulfillment of the

requirements to the Degree of

DOCTOR OF MEDICINE (SIDDHA)

BRANCH I - MARUTHUVAM

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CONTENTS

INTRODUCTION

மருந்தென வேண்டாவாம் யாக்கைக்கு அருந்தியது
அற்றது போற்றி யுணின்.

-குறள்.

Siddha system of medicine is a traditional medicine of Tamilians. It is one of the most antiquated traditional medicine in the world. Siddha system was first described by lord Shiva to his wife Parvathi. She explore all these knowledge to her son lord Muruga. He taught all these knowledge to his disciple sage Agathiyar in Tamil language, so only lord Muruga is called as *Thamizh Kadavul* (God of Tamils).

Siddhars were saints, doctors, alchemists and mysticists all at once. They wrote their findings, in the form of poems in Tamil language, on palm leaves. Sage Agathiyar is considered the guru of all Siddhars. He is the first Siddhar. His disciples and other siddhars contributed thousands of texts on Siddha literatures, including medicine and form the profounder of the system in this world. He is considered as the father of Tamil literature and compiled the first Tamil grammar called Agathiyam. It is believed that he has lived in the 6th or 7th century B.C and specialized in language, alchemy, medicine and spirituality (Yogam and Gnanam).

Siddha is the science of life. According to Siddha philosophy the universe around us is the Macrocosm (Andam) and the human body is considered as the Microcosm (Pindam). Any change in the macrocosm will have its impact in the Microcosm i.e human body. Both are formed by the basic of five elements called as Pancha Boothangal (Panchabootha theory) i.e: Ahayam (Ether), Kattru (Air), Thee (fire), Neer (Water) and Nilam (Earth). These five elements combine to form the three Thathus (Vatha, Pitha, Kaba), the balance of which is very essential for the healthy life.

This holistic ancient science has two objects, viz. to maintain the health of healthy person, and to treat the sick person. The diseases evolving recently in the modern system of medicine (Allopathy) have already been dealt by our tamil inventors, the great Siddhar's. Many millennium backs one such clinical entity is "**Mathumegam**" which is described by "Yoogi Vaithiya chinthamani-800". According to Siddha Maruthuvam Pothu the synonyms of Mathumegam are Neerizhivu, Enippu neer.

"*Tamilians who know about its prominent manifestation of "persistent polyuria" named the disease Megam, Mathu means "honey" (sweetness)****"***.

The term Mathumegam is indicating the idea of sweet substance similar to honey in reset of taste, odor, and color not in concentration, which is secrete profusely through the urinary system.

Diabetes mellitus is recognized as one of the oldest known disease. Historical accounts revel that as early as 700-200 BC. Diabetes mellitus was a well-recognized disease in India. In modern science, Mathumegam is co-relating with Diabetes mellitus. Yoogimuni used the term "Mathumegam" for Diabetes mellitus. Yoogimuni, author of Yoogi Vaithiya Chinthamani-800 has described the signs and symptoms of Mathumegam as "Gunam and Avathaigal". They are may be correlated to sign and symptoms of Diabetes mellitus.

Diabetes Mellitus is a heterogeneous group of metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with the disturbance of carbohydrate, protein and fat metabolism resulting from defects in insulin secretion, action or both. Type 2 Diabetes is one of the major health problems all over the world. According to WHO recent estimates indicate there were 171 million people in the world with diabetes in the year 2000 and this is projected to increase to 366 million by 2030. There were 32 million people with Diabetes in India in 2000, which is projected to rise to 80 million by the year 2030. Increase in prevalence is rapid in urban areas from 2% in 1970s to 12% in 2000 and as well in rural areas.

Diabetes mellitus is one of the cardinal problems in the medical profession because it cannot be cure but some extent controlled. Present days oral anti-diabetic drugs have prominent side effects like hypoglycemia at higher doses, liver problems, diarrhea etc. It is important that efforts are made to develop more efficacious agents with lesser side effects.

The role of medicinal plants in ameliorating the problem of Diabetes is noteworthy because of their low cost, quick positive response and being safe on the body without apparent side effects. Therefore, investigations of these herbal agents for anti-diabetic activity are an important area of research.

In Siddha system of medicine, various herbal, herbo-mineral and minerals are using popularly and very effectively in the treatment of Mathumegam with different disease conditions. Hunting of the Siddha treasures may give valuable information about prevention for the disease, as well as treatment of the disease. This system had got advantage over other medicines in many respects. It aims at treating the patient and the other system aims at treating the disease.

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This study brings out the therapeutic efficacy of siddha polyherbal formulation Atthippattaiyathi kasayam in the treatment of Mathumegam (type-2 Diabetes Mellitus) and to create awareness about the disease, its complications and managements.

AIM AND OBJECTIVE

AIM

The Purpose of this study is to evaluate the therapeutic efficacy of the Siddha polyherbal formulation **ATTHIPPATTAIYATHI KASAYAM** in the treatment of **MATHUMEGAM** as an open clinical trial.

OBJECTIVES

Primary objective:

To evaluate the safety of the Siddha polyherbal formulation **Atthippattaiyathi kasayam** in the treatment of **Mathumegam**

Secondary objective:

1. To Botanical identification and authentication of the trial drug.
2. To prepare the trial drug **Atthippattaiyathi kasayam** as per Siddha literature and analysis of qualitative and quantitative constituents present in the trial drug.
3. To establish the toxicological profile by performing acute oral toxicity studies and sub acute toxicity studies on mice and rats following WHO guidelines.
4. To Study the Safety and efficacy of the test drug through an open clinical trial.
5. To analyze the prevalence of Mathumegam among the society through Age, Sex, Occupation, Distribution etc.

SIDDHA ASPECTS

In siddha system of medicine diseases classified as 4448 types. According to Yoogi Vaithiya Chinthamani Meganoi is classified into 20 types, Mathumegam one among them under pitha type.

தண்மையாய்ச் சலந்தானும் பசுப்பு மஞ்சள்
தானிறங்கு பீசமுங்கோ சமுங்க டுக்கும்
அண்மையாயடிக் கடிக்கு நீரிறங்கும்
அடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையா யடிதனில் தான் பிடிக்கும்
மிக்கான சடம்வெளுத்து மேனி கண்ணும்
பண்மையாய்ப் பஞ்சவாண் டதனிற் கொல்லும்
பகர்கின்ற மதுமியத்தின் பாங்கு தானே.

-யூகிவைத்திய சிந்தாமணி

Definition:

"Mathumegam is a clinical condition characterized by frequent passage of urine more than the normal resulting in deterioration and diminution of Seven Thathus.

சரியான மேகத்தா லபான வாயு
தான்புகைக்கு மேலேறிக் கபாலச் சூடாம்
பெரிதான மேகத்தா லத்தி வெந்து
போமப்பா தசைவெந்து ரத்தம் வற்றிப்
பரிவாகித் தசவாய்வால் மந்தங் கொண்டு
பெருந்தீனி மலபந்தம் உதான வாயு
வரிவாகித் தேகமெலாம் விட நீராலே
மெய்யழிந்து மேகமென்ற திருப தாச்சே

- சித்தர் நாடிநூல்

"Tamilians who know about its prominent manifestation of persistent polyuria named the disease Megam, Mathu means honey (sweetness)".

The term Mathumegam is indicating the idea of sweet substance similar to honey in reset of taste, odour and colour not in concentration, which is excrete profusely through the urinary system.

Synonyms of Madhumegam

According to Siddha Maruthuvam the synonyms of Mathumegam are Enippu neer, Neerizhivu.

Etiology:

The authentic etiological factors described by various siddhars are as follows.

கோதையர் கலவி போதை கொழுத்தமீ னிறைச்சி போதை
பாதுவாய் நெய்யும் பாலும் பரிவுட னுண்பீ ராகில்
சோதபாண் டுருவ மிக்க சுக்கில பிரமே கந்தான்
ஓதுநீ ரிழிவு சேர வுண்டென வறிந்து கொள்ளே

-அகத்தியர் 1200

The above poem quotes that excessive intake of rich food like ghee, fish, milk, toddy and excessive indulgence in sex leads to Mathumegam. The same also discussed in below poem with increased body heat (Pitham) and excessive hunger also leads to Mathumegam.

உட்டிண ரோகத் தாலும் முறும்பெரும் பசியினாலும்
கட்டவிழ் கோதைமாதர் கலவிமட்டிலா மையலாலு
முட்டறா நாலுமாறு மும்மூன்று மொன்று மென்று
திட்டமாய் வருவதென்று திருமமுனி யருளிச் செய்தார்

-அகத்தியர் 1200

The same etiological factor discussed below in Yoogi Vaithiya Chinthamani.

உற்பவிக்கும் பால்நெய்யா லிறைச்சி கள்ளால்
உரிசையாய் மீன்றன்னால் வருவி ருத்த
மற்பவிக்கும் பதார்த்தத் தால்மதுர வஸ்தால்
மந்தங்கள் தனிற்பொசித்தல் வேகாப் பண்டம்
குற்பவிக்குங் குளிந்தவன்ன மங்கை கோசூ்டி
குறித்தநித் திரைதவிர்த லக்கினி மந்தம்
தற்பவிக்குஞ் சரீரந்தான் மிகப்ப ருக்கல்
சஞ்சலந்தான் பயன்படுதல் தரிக்கும் நோயே

இயம்பவே ஆறுகுளம் பின்னஞ் செய்தல்
ஏற்றமாய் பிராமஸ்திரீ சங்கம் பண்ணல்
பயம்பவே பாலகர்களுக் கொளித்துத் தின்னல்
பழமைசலம் போறவழி தனைத்த டுத்தல்
அயம்பவே ஆலயத்திற் சலம்விட் டோர்க்கும்
ஆதியாம் வேதத்தை த்தூசூித் தோர்க்கும்
துயம்பவே சூரியனை வண்ங்கா தோர்கும்
சுருக்காக மேகம்வந் துற்பவிக்குந் தானே

- யூகிவைத்திய சிந்தாமணி

Sexual indulgence:

All Siddhars attribute Mathumegam mainly due to excessive indulgence in sex which results in depletion of total strength of body as a whole, making the individual susceptible to this disease.

கன்னி மயக்கத்தால் கண்டிடும் மேகமே

-நாடிநூல்

கட்டவிழ் கோதைமாதர் கலவிமட்டிலா மையலாலு

-அகத்தியர் 1200

ஸ்திரிபோகம் செய்ததினால் வேவுகொண்டு
சிரசு மட்டும் வெந்துருகிக் கனலே மீறிக்
குறியுடனே மேகந்தான் கொடுமை செய்து
குறைந்து வரும் தாதுவெல்லாம் குன்றிப்போகும்

-குருநாடி

நிறை பூத்த கொங்கையாள் நாயகன் மேகத்தால்
மறை போற்றும் கருப்பத்தில் வளர்ந்தது மேகமே

-திருமூலர்

கிரந்தி புண்ணிரண மேகக் கீசக னென்னுந் துன்மார்க்க
னருந்ததி யென்னும் பாஞ்சாலி யன்னையைக் கண்ணுற்றானே

-தேரன் மருத்துப் பாரதம்

Psychosomatic Factors:

Yoogimuni and other Siddhars also said that, psychosomatic stress resulting in disease like Mathumegam, Gunmam and Kuruthi Azhal.

இயம்பவே ஆறுகுளம் பின்னஞ் செய்தல்
அயம்பவே ஆலயத்திற் சலம்விட் டோர்க்கும்
ஆதியாம் வேதத்தை த்தூகூழித் தோர்க்கும்
துயம்பவே சூரியனை வணங்கா தோர்கும்
சுருக்காக மேகம்வந் துற்பவிக்குந் தானே

- யூகிவைத்திய சிந்தாமணி

Kanma Noi

In the views of Agathiar and Theraiyar, Mathumegam also occurs as a result of bad deeds committed in his or her past. Nowadays it is called as "Genetic factors".

ஆமப்பா மனிதர் செய்த கர்மத்தாலே
அரகரா மேகமென்ற ராசாவாலே
காமப்பா லதினால் பசியுப்ப நாளுங்
கைக்கடங்கா நோய்கள் வரும் கர்மத்தலே

-அகத்தியமுனிவர் கன்மகாண்டம்

தானேபூருவ விதியினால் சாரும் பிணிகளெல்லாம்
மானோர் விழியாள் வேட்கையினால் வருந்தும் பின்னும் பசியால்
தானே பொறுத்து உண்கையினால் தாகந்தன்னால் மிகச்சோர்ந்து
தானே கமலம் புண்ணாகி செய்யும் பிரமேகச் செயல்தானே

-தேரையர்

Classification

Twenty types of Meganoi have been discussed by Yoogimuni, Agathiar and Theraiyar.

வசனித்த மேகமது இரண்டு பத்து
வாததிற் பிறந்தசலம் நாலே யாகும்
பிசனித்த பித்தத்தி லுற்ப வித்த
பேரான சலந்தானு மாறு மாகும்
தெசனித்த சேட்டுமத்தில் உற்ப வித்த
சீரான சலந்தானும் பத்தே யாகும்
இசனித்த இதினுடைய குணாகு ணங்கள்
சுழிவான உற்பத்தி யியம்பக் கேளே

முறையான பித்தசல மாறு மாகும்
முதிர்ந்தஅப் பியமென்றும் பிரமிய மென்றும்
தறையான சாம்பீர்ணம துமிய மென்றும்
சாத்திகமே யாறுவிதந் தன்னாடாறு
மறையான விந்தாறு மேகந் தன்னை
மகதேவர் சொல்லிடவே தேவி கேட்கத்
துறையான குணாகுணத்தை விரித்துச் சொல்ல
சுற்றமாயப் பியத்தின் சுருபங் கேளே.

- யூகிவைத்திய சிந்தாமணி

தக்க தாரனி மானிடத்தோர் கேள்
பக்க மா சலம் வகையுமாமே
நக்க நாயகன் நாயகிக்கே சொல்
மிக்க நந்தி விளம்பி விதித்ததே

கழியும் வாதம் நான்காலும்
கயம் பித்த மாறாலும்
கழியும் சேத்துமம் பத்தாலும்
சொல்லும் நாலஞ்சாய் தோன்றும்
வழியும் வாதம் நான்காமே
மாரு தவிழ்தந் தன்னாலே

-தேரையர் வாகடம்

உட்டிண ரோகத் தாலும் முறும்பெரும் பசியினாலும்
கட்டவிழ் கோதைமாதர் கலவிமட்டிலா மையலாலு
முட்டறா நாலுமாறு முன்மூன்று மொன்று மென்று
திட்டமாய் வருவதென்று திருமமுனி யருளிச் செய்தார்

-அகத்தியர் 1200

According to Yoogi Vaithiya Chinthamani “Meganoi” is classified into 20 types i.e Vatha meganeer- 4, Pitha meganeer - 6 and Kaba meganeer - 10 types. “Mathumegam” comes under Pitham type. Each author who have dealt Meganoi have differently classified them under three doshas and have given names according to their concept. But the number, signs and symptoms are almost identical.

Clinical features

Signs and Symptoms (Gunam and Avathaigal)

Yoogimuni, author of Yoogi Vaithiya Chinthamani has described the signs and symptoms of Mathumegam as “Gunam and Avathaigal".

Gunam

தண்மையாய்ச் சலந்தானும் பசுப்பு மஞ்சள்
தானிறங்கு பீசமுங்கோ சமுங்க டுக்கும்
அண்மையாயடிக் கடிக்கு நீரிறங்கும்
அடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையா யடிதனில் தான் பிடிக்கும்
மிக்கான சடம்வெளுத்து மேனி கண்ணும்
பண்மையாய்ப் பஞ்சவாண் டதனிற் கொல்லும்
பகர்கின்ற மதுமியத்தின் பாங்கு தானே.

கூறான மேகமது இருப துக்கும்
குணந்தனை சிவன்சொல்ல தேவி கேட்க
தாறான தாகமொடு சோக மேகந்
தரியாமல் நீரிழித லிருமல் மூச்சு
ஆறான அருசிசத்தி சித்த பிரமை
அடிக்கடிக்குத் தண்ணீர்தான் ஆங்கே கேட்கல்
ஈறான இடுப்புக்குள் கடுப்பு காணல்
எலும்புழற்ற லழற்றலோ டெரிவுண் டாமே

எரிவோடு சரீரமெல்லா மறைபட் டாற்போல்
எழிலுடம்பு நோதல்நித் திரையில் லாமை
வரிவோடு வாய்வுமெத் தவும்ப றிதல்
மனதுசஞ் சலப்படுதல் காற்று வேண்டல்
மெரிவோடு மேல்மூச்சு மிகவுண் டாதல்
விக்கலோடு மயக்கந்தான் மெத்தக் காணல்
தெரிவோடு தேகமெங்கும் வெளுருண் டாதல்
தேகமெத்த வாலோபப் படுதல் காணே.

-யூகிவைத்திய சிந்தாமணி

Passing of urine in large quantity at frequent intervals, while passing urine the patient experience burning and spasmodic pain in the urethra and dull pain in the testis. The urine has yellow color, and produces white sediments which adhere to the bottom of the vessel in which it is collected. The skin is pale and there is generalized body pain. If it is not treated in time resulting in death within five years of period.

இனிப்பான இனிப்பல்ல ஈ வந்தாடும்
ஒரு துளிவாய் விட்டார்கைப் பிணியாய் தோன்றும்

-குருநாடி

The above description quotes that ant and flies are attracted to the site of voided urine and when the urine is heated it gives honey odour.

Avathaigal

காணவே முதலவத்தை சரீரந் தானும்
கனமாகப் பருத்திறுகு நீர்த்து வாரம்
வேணவே வெண்டாக்கி யகலம் பண்ணும்
மிக்கவிரண் டாமவத்தை விளம்பக் கேளாய்
மூணவே மூத்திரப் பீடையுமாச் சுக்கில
முகமழுகித் தேஜசுதான் மிகவே குன்றும்
நாணவே மூன்றாகு மவத்தைக் குந்தான்
நாவரளும் வாயுவது மீறுந் தானே.

தானான நாலவத்தை யங்க தாகம்
சன்னியது பாதமுண்டா மைந்த வத்தை
தேனான நீர்பெருகுந் தாது நசுஷ்டம்
நிலையாறா மவத்தையுடற் கிடைகொள் ளாது
மூனான மூர்ச்சைவரு மேழ வத்தை
மிக்கவரோ சிகஞ்சுவாசந் தேக சாட்யம்
ஏனான எட்டாவ தவத்தை தானே
எழுகிரந்தி பிளவையுந்தான் மிகவுண் டாமே.

உண்டாகு மொன்பதா மவத்தை கேளாய்
ஒழுக்கான ஆசாரங் கிருமி யுண்டாம்
மண்டான பத்தாந்தா னவத்தை கேளாய்
பாரமாம் கூடியங்கண்டு பரத்துக் கேகும்
விளங்கியதோர் தசவவத்தை விபரஞ் சொன்னோம்.

-யூகிவைத்திய சிந்தாமணி

The below signs and symptoms are occurring in undiagnosed and improperly treated cases.

1. First symptoms of Megam disease are obesity and dilation of urethral.
2. Body becomes dry and loses its lusture due to excessive secretion and flow of urine mixed with vital fluid (semen).
3. Dryness of the tongue and distension of abdomen due to formation and accumulation of excessive gas.
4. Delirium (Toxic condition) supervenes following dehydration due to excessive elimination of tissue fluid.
5. Restlessness due to loss of vital fluid in urine.
6. Breathlessness and restlessness**.**
7. Nausea, tastelessness, laboured breathing, exhaustion.
8. Carbuncle and multiple abscess formation.
9. Maggot formation and generalized emaciation.
10. Intractable troublesome, cough with profuse expectoration leading to death.

PINIYARIMURAIMAI (Diagnosis)

It is very important part of the treatment. It is helpful to select the correct line of treatment and good prognosis. It is based upon the following diagnostic methods.

- Poriaal therthal
- Pulanaal arithal
- Vinadhal
- Envagai thervugal

Poriaal therthal

The physician should examine the patient's porigal.

- Mei: Feels all types of sensations
- Vaai: For knowing taste
- Kan: Meant for vision
- Mooku: For knowing the smell
- Sevi: For hearing

Pulanaal Arithal:

The physician should examine the patient's pulangal (Functions of the sensory organs)

- Oosai - Perception of sound
- Ooru - Perception of sensation
- Oli - Perception of vision
- Suvai - Perception of taste
- Nattram - Perception of smell

Vinadhal (Interrogation)

The physician should interrogate about the patient's name, age, occupation, native, socio- economic status, dietetic habits, prone to any allergens, complaints, history of previous illness, history of past illness, family history and frequency of attacks. If the patient is unable to speak, or is a child physician should interrogate the details with his immediate relatives who are taking care of him.

Ennvagai thervugal

The prime method adopted to diagnose the disease is by means of "Ennvagai thervugal". The value of ennvagai thervugal is very important for diagnosing purposes, which is the unique and special method described in siddha system of medicine.

1. Naadi
2. Sparisam
3. Naa
4. Niram
5. Mozhi
6. Vizhi
7. Malam
8. Moothiram

Eight different kinds of tests to be applied or attended by a physician before arriving a correct diagnosis. Envagai thervukal is considered as Physician's Instruments.

"நாடி பரிசம் நா நிறம் மொழி விழி
மலம் மூத்திரம் இவை மருத்துவராயுதம்"
-தேரையர்

1. Naadi (Pulse)

Naadi is the vital force. Any change in the three dhoshas are best diagnosed by feeling the nadi. Naadi is an important observation for diagnosis and prognosis. Naadi is responsible for the existence of life and can be felt one inch below the wrist on the radial side by means of palpation with the tips of index, middle and ringfinger corresponding to Vatham, Pitham and Kabam.

Site and procedure to feel Naadi according to Agasthiyar

"கரிமுக னடியை வாழ்த்திக்
கைதனில் நாடி பார்க்கில்
பெருவிரலங்குலத்தில்
பிடித்தடி நடுவே தொட்டால்
ஒருவிரலோடில் வாதம்
உயர் நடுவிரலிற் பித்தம்
திருவிரல் மூன்றி லோடில்
சிலேத்தும நாடி தானே".
-அகத்தியர் நாடி

According to Thirumoolar, the ten sites for feeling Naadi

தாது முறைகேள் தனித் தகுதிச் சந்தோடு
ஓதுறு காமிய முந்திநெடு மார்பு
காது நெடுமூக்கு கண்டம் கரம்புருவம்
போதுறுமுச்சி புகழ் பத்தும் பார்த்திடே

-திருமூலர் நாடி

Formation of Naadi

Naadi	+	**Vayu**	=	**Uyir Thathu**
Idakalai	+	Abanan	=	Vatham
Pinkalai	+	Pranan	=	Pitham
Suzhumunai	+	Samanan	=	Kabam

Normally the three humors Vatham, Pitham and Kabam exist in the ratio 1: ½ : ¼ The derangement in these ratio leads to various disease entities and is best diagnosed by feeling the Naadi.

Naadi in Madhumegam

“இருமியே பித்தமும் வாதமும் கூடில்
மருவுசல மேகம் வாருதி போலாகும்
உருவம் வேறாகு முண்டவுடற் காய்ந்திடும்
உருகவே வூனோடு உறிஞ்சி இனிக்குமே”

-திருமூலர் நாடி

The above stanza describes that excessive elimination of urine containing sugar are always primarily due to combined vitiation of Vatha, and Pitha functional factors in the body. The pitha and vatha vitiation is indicated clinically by excessive hunger, thirst, over-eating, emaciation and passing of large quantities of urine.

“இனிக்கின்ற வாத்திடை சேரில் ஐயந்தான்
பனிக்கின்ற கள்ளுப் பதனிபோல் நீரோடும்
கனக்கின்ற மேனிகரைந்து வெளுப்பேறும்
கனிக்குமது மேகந் தப்பாதே ஐயமும்”

-திருமூலர் நாடி

The above poem indicates that initially vatha and kabam get deranged leading to vitiation of pitha thathu finally. When the aggravated vatha naadi combines with

aggravated kaba naadi, there is genesis of megam disease in the body. The meganeer thus formed and eliminated has the consistency and appearance of toddy. The affected individual's body is pale and emaciated. This is the typical clinical picture of Mathumegam.

“துரணமுடன் நீர்ப்பாடு கெர்ப்பப் பாடானாற்
சொல்லுகிறேன் நாடியெல்லாங் கழன்று காணும்”

-பரிபூரண நாடி

In the above lines, it is said that all the three naadi are feeble and weak in Mathumegam.

“பற்பிடித்த மேகம் என்றால் பித்தமீறும்
பாலகனே காங்கை கொண்டு நீராம் பாரே”

-பரிபூரண நாடி

By the above lines it is clearly stated that aggravation of pitha naadi results in increased udal kaangai. Eventually this leads to emaciation of seven udalthathus resulting in Meganeer.

“நீர்மேகமானவர்க்கு நாடி காணும்
நிர்ணயமாய் நாடியெல்லாம் பெலமே கெட்டுக்
கார்மேகம் போல வந்த எரியின் மேலே
கண்டு விழும் புழுப்போலவே புரண்டு காணும்”

-பரிபூரண நாடி

All the three naadi are felt feeble in those suffering from Neerizhivu Noi. The character of the pulse is compared to that of wriggling movements of a worm that has fallen into the fire.

2. Sparisam (Palpation)

The following points are elicited by Sparisam, the temparatue of skin (heat or cold), smoothness, roughness, softness, sweat, dryness.

3. Malam (Faeces)

In the examination of Malam, Niram (Colour), Nurai (Froth), Erugal (Solid), Elagal (Semisolid or liquid), quantity (increased or decreased) and smell can be noted. Other quantitative analysis such as, presence of blood, mucus, undigested matter in the stools and odour can also be studied.

4. Moothiram (Urine)

In the examination of urine, colour, odour, quantity of urine, the presence of froth, deposits, blood, pus, inorganic sediments, abnormal constituents such as sugar, protein etc, and the frequency of micturition, flow pattern, burning sensation if any while passing urine are to be noted.

The diagnosis and prognosis are usually arrived at by methods of urine examinations as follows,

i) Neerkuri

ii) Neikuri

Collection of Urine

“அருந்துமாறிரதமும் அவிரோதமதாய்
அஃகல் அலர்தல் அகாலவூண் தவிர்ந்தழற்
குற்றள வருந்தி உறங்கி வைகறை
ஆடிக் கலசத் தாவியே காது பொய்
தொரு முகூர்த் த்க்கலை குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே”

- தேரர் நீர்குறி நெய்குறி

Prior to the day of urine examination, the patient should be advised to take a balanced diet and should have good rest. The first voided urine of the patient is collected in a glass container. The collected urine is subjected into neerkuri and neikuri examination within1½ hours and the following are to be observed colour, smell, frothy, volume, specific gravity and sedimentation and the shape of the oil spread in the urine.

i) Neerkuri

“வந்த நீர்க்கரி எடை மணம் நுரை எஞ்சலென்
றைந்தியலுளவவை யறைகுது முறையே”

- சித்தமருத்துவாங்க சுருக்கம், ப.எண்:510

In Neerkuri Niram, Edai, Manam, Nurai and Enjal of the urine voided is noted. This has been already mentioned in Envagai thervugal. The urine should be examined only according to the rules and regulations but at time of emergency they can be relaxed.

Niram : It indicates the colour of urine voided.

Edai : It indicates the specific gravity of urine (increased or decreased quantity).

Manam : In indicates the smell of urine voided.

Nurai : It indicates the frothy nature of urine voided.

Enjal : It indicates the quantity of urine.

Neerkuri of Mathumegam is studied as follows:

Niram : Clear and white. This is due to Kaba vitiation and it is not amenable to treatment.

Edai : Urine is thick and its consistency is like honey.

Manam: Smells like honey. Ants and flies are attracted towards the voided urine. It indicates that it contains some sweet substances which attract the ants and flies.

Nurai : It is frothy at the time of urination.

Enjal : Large quantity of urine is passed. This will result in the loss of large Volume of water and life sustaining minerals resulting in fatigue, exhaustion and weakness.

If the urine is lightly transparent, it indicates the vitiation of kaba in which the prognosis is said to be very bad. The above findings of Neerkuri in Madhumegam is described in Siddha texts are as follows.

“தண்மையாய்ச் சலந்தானும் பசுப்பு மஞ்சள்
தானிறங்கு பீசமுங்கோ சமுங்க டுக்கும்
அண்மையாயடிக் கடிக்கு நீரிறங்கும்
அடிக்கடிக்கு அரைநாழி தனிலே காணும்
வெண்மையா யடிதனில் தான் பிடிக்கும்
மிக்கான சடம்வெளுத்து மேனி கண்ணும்
பண்மையாய்ப் பஞ்சவாண் டதனிற் கொல்லும்
பகர்கின்ற மதுமியத்தின் பாங்கு தானே”.

-யூகிவைத்திய சிந்தாமணி

The description of the physical feature of the Mathumegam urine in siddha system agree with the description of the physical feature of diabetic urine by modern science. The name of the disease itself indicates that the urine passed contains a substance which is not only sweet but also emanates the odour of honey.

Neikuri

A drop of gingely oil is dropped into a wide vessel containing the urine to be tested and kept under the sunlight in a silent place and observed for one minute. The variations of the three thathus in the disease condition studied and the prognosis of the disease can be observed from the spreading pattern of gingely oil in the urine surface.

“அரவென நீண்டினஃதே வாதம்”
-நோய்நாடல் நோய்முதல் நாடல் திரட்டு

The drop of oil lengthening like a snake indicates Vatham.

“ஆழிபோற் பரவின் அஃதே பித்தம்”
-நோய்நாடல் நோய்முதல் நாடல் திரட்டு

The drop of oil spreading like a ring it indicates Pitham.

“முத்தொத்து நிற்கின் மொழிவதன் கபமே”
-நோய்நாடல் நோய்முதல் நாடல் திரட்டு

The drop of oil look like a pearl shape it indicates kabam.

Thinai (Land or Place)

Nilam is classified into five types depending on the flora and fauna, landscape and ecology study of five places is very much necessary as some of the diseases are more prevalent in a particular land.

- **Kurinji**: Mountain and its surroundings. Liver diseases are common.
- **Mullai:** Forest and its surroundings Pittha diseases, Vadha diseases are common.
- **Marutham**: Paddy field and its surroundings. This is the ideal place for healthy living.
- **Neithal:** Sea and seashore. Liver diseases occuring in combination with other diseases.
- **Palai**: Desert and its surroundings. Vatha, Pittha and Kabha diseases occur

Nowadays Mathumegam is prevalent in all five types of lands due to sedentary lifestyle.

Paruvakalam (Season)

In siddha system of medicine Siddhars have classified the seasons into six each comprising of two months.

Sl.No.	Kalam	Kuttram	State of Kuttram
1	Kar kalam (Avani & Purattasi) (Aug. 17 - Oct 17)	Vatham Pitham	Vettrunilai Valarchi Thannilai valarchi
2	Koothir Kalam (Iypasi & Karthigai) (Oct. 18 - Dec. 15)	Vatham Pitham	Thannilai Valarchi Vettrunilai valarchi
3	Munpani Kalam (Maragazhi & Thai) (Dec. 16 - Feb. 12)	Pitham	Thannilai Adaithal
4	Pinpani Kalam (Masi & Panguni) (Feb.13 - Aprl. 13)	Kapham	Thannilai valarchi
5.	Elavenir kalam (Chithirai & Vaikasi) (Aprl. 14 - June 14)	Kapham	Vettrunilai valarchi
6.	Mudhu venir Kalam (Aani & Aadi) (June 15 - Aug. 16)	Vatham Kapam	Thannilai Valarchi Thannilai adaithal

Mukkuttram

1. Vatham
2. Pitham
3. Kabam

I. Vatham

Vatham is the kinetic energy which influences all motions. It denotes Vayu dryness, pain, flatulence. It is classified into 10 types based on functions.

Location of Vatham in our body

Vatham is located in abanan, Faeces, Idakalai, Spermatic cord, Nerve plexus, Joints, Hair follicles and Muscles, Bones and Thigh. Increase or decrease of Vatham can cause some standing symptoms which are below,

Increase

Pain in the body, twitching & piercing pain, inflammation, reddish complexion, roughness of skin, hardness of limbs, astringent sense of taste in the mouth, taste not palatable, sweating during sleep, traumatic pain, constipation, oliguria, blackish discolouration of skin,stool, urine and muddy conjuctiva, tremors, abdomen distention, insomnia, and breathlessness.

Decrease

Body pain, feeble voice, diminished competence of intellectual functions and syncope etc.

Types of Vatham

1. Piranan (Uyir Kaal)

It is mainly responsible for respiration and it is necessary for proper digestion and control knowledge.

2. Abanan (Keezhnokku Kaal)

It is responsible for voiding of urine, stools, semen and menstrual flow.

3. Viyanan (Paruvkaal)

It is used to feel all types of Sensations. It carries nutrients to all over the body flexes and extends the movable joints.

4. Uthanan (Maelnokku kaal)

Responsible for all kinds of upward motion such as nausea, vomiting and eruption.

5. Samanan (Nadukkaal)

Considered essential for proper digestion assimilation and carries the digested nutrients to each and every organ.

6. Nagan Helps in opening and closing of the eyes.

7. Koorman Responsible for yawning, vision and lacrimation.

8. Kirugaran Responsible for salivation, nasal secretion and appetite.

9. Dhevathathan

Induces and stimulates a person to become alert, get anger, to quarrel, to sleep, to become lazy etc.

10. Dhananjeyan

Resides in the cranial cavity and produces bloating of the body after death. This leaves from the body after 3 days forming a way through the skull bone.

In case of Madhumegam

1.Abanan - Affected (increased volume and frequency of micturation).

2.Viyanan - Affected (altered sensation).

3.Udhanan - Affected (nausea, vomiting).

4.Koorman - Affected (Blurring of vision)

5.Kirugaran - Affected (Polydipsia).

The above mentioned types of vatham are affected in mathumegam.

II. Pitham

It is the thermal life force of the body. Pitha in the body is followed by the derangement of metabolic energy caused by the involvement of Vatha.

Location of Pitham in our body

Pingalai, Piranan, Urinary bladder, Heart, Moolakkini, Head, Abdomen, Sweat, Blood, Saliva and Digested material etc.

Increase or decrease of Pitham can cause some standing symptoms which are below

Features of increased pitham:

Yellowish discolouration of eyes, skin, urine and motion. Polyphagia, polydipsia, burning sensation all over the body, sleeplessness, acidity, profuse sweating and dizziness etc.

Features of decreased pitham:

Loss of appetite, cold, pallor, symptoms associated with defective growth of Kapham.

Types of Pitham

1. Anar Pitham

It peps up the appetite aids in digestion.

2. Ranjaga Pitham

It is responsible for the colour and contents of the blood.

3. Pirasaga Pitham

It gives Complexion of the Skin.

4. Sathaga Pitham

It is Necessary to carry out regular works properly.

5. Aalosaga Pitham

It is responsible for the perception of Vision**.**

In case of Mathumegam

1. Anar Pitham

Affected – Polyphagia

2. Alosaga Pitham :

Affected - Blurring of Vision.

III. Kabam

It is responsible for the stream lined functions of the body and body's defence mechanism to be intact.

Location of Kabam in our body

Samanan, Suzhumunai, Vinthu, Head, Fat, Marrow, Nose, Colon,Joints etc.

Increase or decrease of Kabam can cause some standing symptoms which are below,

Features of increased kabam:

Loss of appetite, excessive salivation, heaviness, excessive musculature, dyspnoea, excessive sleepiness, fair complexion, itching, dullness, cold, loss of sensation, sweetness in mouth and indigestion etc.,

Features of decreased kabam:

Prominence of bony edges, dry cough, lightness, profuse sweating and palpitation.

Types of Kabam

1. Avalambagam

Lies in the respiratory organs, exercises authority over other Kabams and controls heart and circulatory system.

2. Kilethagam

It is found in stomach as its seat moistens the food, softens and helps it for digestion.

3. Pothagam

Tongue is the centre for pothagam, it is responsible for the sense of taste.

4. Tharpagam

Head is the centre for tharpagam, it gives cooling to the eyes.

5. Santhigam

It lies in the joints and is responsible for the lubrication and true movements of joints.

In the case of Mathumegam

1. Kilethagam - Gets affected due to increased appetite.

Udal Kattugal

These are seven basic principles which constitute the entire body. There are seven Udal Kattugal described in Siddha text.

1. Saram

It strengthens the body and mind.

2. Senneer

It is responsible for the nourishment, Strength, Vigour and healthy complexion.

3. Oon

It gives structure and shape of the body and is responsible for the movement of the body.

4. Kozhuppu

It helps for lubrication of joints and other parts of the body to facilitate their functions.

5. Enbu

It supports the body structure and protects the organs. It is responsible for the posture and movement of the body.

6. Moozhai

It nourishes the bone marrow.

7. Sukkilam/ Suronitham

It is responsible for reproduction.

Udal Kattugal	**Increased features**	**Decreased features**
1) Saaram	Leads to a disease identical to the increase in kabam like loss of appetite ,profuse salivation, depression etc.,	Dryness of the skin diminished activity of the sense organs, lassitude, Loss of weight, Intolerance to sounds.
2) Senneer	Increased blood pressure, boils in eye brow, scalp neck, lips and legs, skin jaundice, haemaeturia, Colic pain	Eagerness to sore an foods. Tiredness, lassitude,d cold anaemia.
3) Oon	Deposition of fat around the neck, face, abdomen, thigh, genitalia, etc.,	Muscle wasting, tiredness.
4) Kozhuppu	Identical feature of increased oon associated with dyspnoea on exertion	Loin pain, emaciation splenomegaly
5) Enbu	Excessive ossification and dentition	Pain in joints, loss of hair, extraction of foot, weak bone and nail.
6) Moozhai	Weariness of the body and eye, swollen interphalangeal joints, oliguria and non healing ulcer.	Osteporosis and shunken eyes
7) Sukkilam (or) Suronitham	Increased sexual activity urinary calculi etc	Pain in the genitalia, failure to production.

In case of Mathumegam all seven thathus are affected.

1. Saram- Tiredness
2. Senneer- Reduced Strength
3. Oon- Weight loss
4. Kozhuppu- Weight loss (or) obese
5. Enbu - Joint pain
6. Moolai - Affected
7. Sukkilam- Body becomes dry and loses its lusture due to excessive flow of urine mixed with vital fluid.

In the case of Mathumegam frequent passage of increased amount of urine results in gradual diminition of seven thathus.

“சரியான மேகத்தா லபான வாயு
தான்புகைக்கு மேலேறிக் கபாலச் சூடாம்
பெரிதான மேகத்தா லத்தி வெந்து
போமப்பா தசைவெந்து ரத்தம் வற்றிப்
பரிவாகித் தசவாய்வால் மந்தங் கொண்டு
பெருந்தீனி மலபந்தம் உதான வாயு
வரிவாகித் தேகமெலாம் விட நீராலே
மெய்யழிந்து மேகமென்ற திருப தாச்சே”

- சித்தர் நாடிநூல்

In this poem Oon and Senneer were affected.

Noi Kanippu Vivaadham (Differential Diagnosis):

- **Thelineer**

The signs and symptoms of the thelineer were polyuria (voided urine is clear and snow like appearence), polydypsia, loss of appetite, loss of body weight, dryrness of skin, constipation or diarrhoea, muscle cramps. Due to the presence of loss of appetite and absence of excessive sugar in blood and urine sample, this disease can be differentiated from mathumegam.

- **Due to prolonged intake of diuretics**

Due to the history of prolonged intake of diuretics the patient may have symptoms of polyuria. But due to the absence of excesssive sugar in blood and urine sample the symptoms can be differentiated from mathumegam.

Noinidhanam (Prognosis):

As per Siddha system four types of Meganeer formed as a result of Vatham is incurable. The six types arising due to the vitiation of pitham could be cured with difficulty. But then ten types of meganeer arising due to Iyyam are curable by proper treatment.

Line of Treatment:

The aim of Noi Neekkam is based on

1. Treatment of the disease by internal medicines
2. Diet and advices
3. Yoga therapy

The line of treatment is described as follows,

In Theran Maruthuva bharatham, it is said that the disease has been caused by excessive sexual indulgence. Excessive sexual indulgence leads to the formation of Megam which gradually affect all the seven thathus and finally sets in the genitourinary system resulting in excessive excretion of urine, tasting sweet as honey.

“கிரந்தி புண்ணிரண மேகக் கீசக னென்னுந் துன்மார்க்க
னருந்ததி யென்னும் பாஞ்சாலி யன்னையைக் கண்ணுற்றானே”

-தேரன் மருத்துப் பாரதம்

In Theran Maruthuva bharatham, Megam is alluded to Keesagan and Mathumegam is alluded to Sainthavan, are also alluded to certain metals namely Bheeman for Rasam (Mercury), Dharmar for Ayam (Iron) and other brothers for Steel, Silver, Gold, Lead and Copper.

So, Parpam and chendooram of above metals should be used one by one with suitable Anubanam for the treatment of Meganeer especially Mathumegam.

Treatment:

Siddhars aimed at bringing the three doshas in equilibrium in the treatment of disease. Siddhars prescribed a minimum dosage initially and then increased the dose gradually.

“வேர்பாரு தழைபாரு மிஞ்சினக்கால் மெல்ல மெல்ல
பற்ப செந்தூரம் பாரே”

So, metal and mineral preparations like Parpam and Chendooram are followed by Herbal preparations like Kudineer, Chooranam and Ilagam. There are thousands of preparations for Mathumegam and its complications found in various Siddha text books like Kudineer, Chooranam, Ilagam, Parpam and Chendooram, etc.

Diet and advices:

தவிக்க வேண்டியவை:

- சர்கரை, தேன், பனைவெல்லம், இனிப்பு வகைகள், மிகு இனிப்பான பழங்கள், பழச்சாறு வகைகள், கிழங்கு வகைகள், நெய், பால், தயிர், எண்ணெய் பலகாரங்கள், கோழி, ஆடு, முட்டை மஞ்சள்கரு ஆகியவற்றை தவிர்கவும்.
- மது, புகை, வெற்றிலை, பாக்கு இவற்றை தவிர்கவும்.

சேர்க்க வேண்டியவை:

- தானிய கலவை, முழைகட்டிய தனியம், பருப்பு வகைகள், பச்சடி வகை, சோயாபீன் ஆகியவை முக்கிய உணவுகள் ஆகும்.
- பச்சை இளம்காய்கள், நார்சத்துக் காய்கள், கீரைகள் மற்றும் மோர், கடல்மீன், கொழுப்பற்ற மாமிசம் ஆகியவற்றிலிருந்து புரதங்களை பெறுவது நன்று.
- முழுதானியங்கள், முழுபருப்பு வகைகள், சோயாபீன், பச்சை இலைக்காய்கறிகள் மற்றும் வெந்தயம் ஆகியவை நார்சத்துமிக்க உணவுகள்.
- கடலை எண்ணெய், நல்லெண்ணெய், பருத்திவிதை எண்ணெய், ரைஸ்பிரன் எண்ணெய், சாபிளார் எண்ணெய் ஆகிய எண்ணெய் வகைகளை பயன்படுத்தவும்.
- தினசரி 6கி உப்பு மட்டுமே எடுத்துக்கொள்ளவும், ஊறுகாய், அப்பளம், சட்டினி மற்றும் உப்பில் ஊறிய தயாரிப்பு உணவுகளை தவிர்கவும்.
- தினசரி 45 நிமிடம் வேக நடை நடப்பது நன்று.

Yoga Theraphy

Yoga is India’s unique contribution to the world. The word “Yoga” is derived from the Sanskrit word “yuj” which means bind, join, or attach. Yoga therefore is an art which brings an incoherent and scattered mind to a reflective and coherent state.

Yogasanaas are nothing but a kind of Yogic exercises. There are innumerable types of Aasanaas. According to Thirumoolar,

“இயம னியமமே எண்ணிலா ஆதனம்
நயமுறு பிராணயாமம் பிரத்யாகார
சயமிக தாரணை தியானசமாதி
அயமுறும் அட்டாங்க மாவது மாமே”

-திருமூலர்

Each Yogasanam is indicated for a definite effect in a particular region of the system by stimulating the internal organs to function in a normal way and to co-ordinate bodily functions. Villaasanam and Mayuraasanam are specifically helps in the treatment of Mathumegam.

- In Mayuraasanam the presence of conjoined elbow against umbilicus region activates the pancreas to work more.
- In Villaasanam the whole abdominal organs including the pancreas are properly tuned and stimulated well by the increase of intra abdominal pressure motivated towards pancreas.

It has been proved that Aasanaas are useful in regulating the pancreas, but in practice the physician should bear in his mind whether in a particular case Yoga alone can be useful or a combined drug administration is also essential.

The following Aasanas are advised for controlling Madhumegam

- Chakkaraasanam
- Mathsyaasanam
- Pachimothaasanam
- Pujangaasanam
- Padmaasanam
- Sarvaangaasanam

All these Aasanas should be practiced daily and regularly which can be of immense value to patients of Madhumegam. All these Aasanas activate the pancreatic cells and have a curative value. These help in restoring cellular function of the pancreas and activate them to work more.

MODERN ASPECTS

Definition

Diabetes mellitus is a metabolic-cum-vascular syndrome of multiple etiologies characterized by chronic hyperglycemia with disturbance of carbohydrate, fat and protein metabolism resulting from defects in insulin, insulin action or both. This disorder is frequently associated with long term damage, which can lead to failure of organs like eyes, kidneys, nerves, heart and blood vessels.

Epidemiology

In recent years, India has witnessed a rapid exploding epidemic of diabetes. Indeed, India today leads the world with its largest number of diabetic people in any given country. WHO estimates that there are 32 million people with Diabetes in India in 2000, which is projected to rise to 80 million by the year 2030. Increase in prevalence is rapid in urban areas from 2% in 1970s to 12% in 2000 and in rural areas also it is now beginning to increase.

Epidemic of Type 2 Diabetes

There are two main form of diabetes. Type 1 diabetes (insulin dependent) is primarily due to autoimmune-mediated destruction of pancreatic beta cells, resulting in absolute insulin deficiency. While type 1 diabetes is also on the increase the actual numbers of people with type 1 diabetes in india is relatively speaking still small. Type 2 diabetes (non-insulin dependent) on the other hand accounts for over 90-95% of all diabetic people and is characterized by insulin resistance and/or abnormal insulin secretion, either of which may predominate. The diabetes epidemic particularly to type 2 diabetes and is taking place both in developed and developing nations with particular reference to India and is predominantly due to the changing demography and increased longevity.

ANATOMY OF THE PANCREAS:

The pancreas is a compound racemose gland, analogous in its structures to the salivary glands, though softer and less compactly arranged than those organs. Its secretion, the pancreatic juice, carried by the pancreatic duct to the duodenum, is an

important digestive fluid. In addition the pancreas has an important internal secretion, probably elaborated by the cells of Langerhans, which is taken up by the blood stream and is concerned with sugar metabolism. It is long and irregularly prismatic in shape; its right extremity, being broad, is called the head, and is connected to the main portion of the organ, or body, by a slight constriction, the neck; while its left extremity gradually tapers to form the tail. It is situated transversely across the posterior wall of the abdomen, at the back of the epigastric and left hypochondriac regions. Its length varies from 12.5 to 15 cm, and its weight from 60 to 100 gm.

Microscopic anatomy of islets of Langerhans

They are found more in the tail of the pancreas than in the other parts. They form about 1 – 2% of pancreatic weight. There are about 2 millions of islets in human pancreas. Each islet has an epithelial mass, tunneled by labyrinthine capillaries. The position of the islets is mostly within the lobules, rather than between them. Each spheroid islet is surrounded by reticular membrane. Islet tissue is arranged in irregular anastomosing cellular plates. Their epithelial cords are separated by blood vessels. A sphincter controls the blood supply. The histological structure of the islets shows Alpha, Beta and Delta cells.

Beta cells are the source of insulin hormone. The cells are polyhedral, the nuclei are centrally or eccentrically placed, the cytoplasm is grannular, filled with prominent secretary vacuoles containing few ribosomes. The secretory granules show species variations. In man they are spherical or elongated crystalline body.

Insulin

Biosynthesis

Insulin is produced in the beta cells of the pancreatic islets. It is initially synthesized as a single-chain 86-amino-acid precursor polypeptide, preproinsulin. Subsequent proteolytic processing removes the aminoterminal signal peptide, giving rise to proinsulin. Proinsulin is structurally related to insulin-like growth factors I and II, which bind weakly to the insulin receptor. Cleavage of an internal 31-residue fragment from proinsulin generates the C peptide and the A (21 amino acids) and B (30 amino acids) chains of insulin, which are connected by disulfide bonds. The mature insulin

molecule and C peptide are stored together and cosecreted from secretory granules in the beta cells. Because the C peptide is cleared more slowly than insulin, it is a useful marker of insulin secretion and allows discrimination of endogenous and exogenous sources of insulin in the evaluation of hypoglycemia. Pancreatic beta cells cosecrete islet amyloid polypeptide (IAPP) or amylin, a 37-amino-acid peptide, along with insulin. The role of IAPP in normal physiology is unclear, but it is the major component of the amyloid fibrils found in the islets of patients with type 2 diabetes, and an analogue is sometimes used in treating both type 1 and type 2 DM. Human insulin is now produced by recombinant DNA technology; structural alterations at one or more residues are useful for modifying its physical and pharmacologic characteristics.

Secretion

Glucose is the key regulator of insulin secretion by the pancreatic beta cell, although amino acids, ketones, various nutrients, gastrointestinal peptides, and neurotransmitters also influence insulin secretion. Glucose levels > 3.9 mmol/L (70 mg/dL) stimulate insulin synthesis, primarily by enhancing protein translation and processing. Glucose stimulation of insulin secretion begins with its transport into the beta cell by the GLUT2 glucose transporter. Glucose phosphorylation by glucokinase is the rate-limiting step that controls glucose-regulated insulin secretion. Further metabolism of glucose-6-phosphate via glycolysis generates ATP, which inhibits the activity of an ATP-sensitive K^+ channel. This channel consists of two separate proteins: one is the binding site for certain oral hypoglycemics (e.g., sulfonylureas, meglitinides); the other is an inwardly rectifying K^+ channel protein. Inhibition of this K^+ channel induces beta cell membrane depolarization, which opens voltage-dependent calcium channels (leading to an influx of calcium), and stimulates insulin secretion. Insulin secretory profiles reveal a pulsatile pattern of hormone release, with small secretory bursts occurring about every 10 min, superimposed upon greater amplitude oscillations of about 80–150 min. Incretins are released from neuroendocrine cells of the gastrointestinal tract following food ingestion and amplify glucose-stimulated insulin secretion and suppress glucagon secretion. Glucagon-like peptide 1 (GLP-1), the most potent incretin, is released from L cells in the small intestine and stimulates insulin secretion only when the blood glucose is above the fasting level. Incretin analogues, such as exena-tide, are being used to enhance endogenous insulin secretion.

Action

Once insulin is secreted into the portal venous system, ~50% is degraded by the liver. Unextracted insulin enters the systemic circulation where it binds to receptors in target sites. Insulin binding to its receptor stimulates intrinsic tyrosine kinase activity, leading to receptor autophosphorylation and the recruitment of intracellular signaling molecules, such as insulin receptor substrates (IRS) IRS and other adaptor proteins initiate a complex cascade of phosphorylation and dephosphorylation reactions, resulting in the widespread metabolic and mitogenic effects of insulin. As an example, activation of the phosphatidylinositol-3'-kinase (PI-3-kinase) pathway stimulates translocation of glucose transporters (e.g., GLUT4) to the cell surface, an event that is crucial for glucose uptake by skeletal muscle and fat. Activation of other insulin receptor signaling pathways induces glycogen synthesis, protein synthesis, lipogenesis, and regulation of various genes in insulin-responsive cells.

Glucose homeostasis reflects a balance between hepatic glucose production and peripheral glucose uptake and utilization. Insulin is the most important regulator of this metabolic equilibrium, but neural input, metabolic signals, and other hormones (e.g., glucagon) result in integrated control of glucose supply and utilization. In the fasting state, low insulin levels increase glucose production by promoting hepatic gluconeogenesis and glycogenolysis and reduce glucose uptake in insulin-sensitive tissues (skeletal muscle and fat), thereby promoting mobilization of stored precursors such as amino acids and free fatty acids (lipolysis). Glucagon, secreted by pancreatic alpha cells when blood glucose or insulin levels are low, stimulates glycogenolysis and gluconeogenesis by the liver and renal medulla. Postprandially, the glucose load elicits a rise in insulin and fall in glucagon, leading to a reversal of these processes. Insulin, an anabolic hormone, promotes the storage of carbohydrate and fat and protein synthesis. The major portion of postprandial glucose is utilized by skeletal muscle, an effect of insulin-stimulated glucose uptake. Other tissues, most notably the brain, utilize glucose in an insulin-independent fashion [Harrison's Principle of Internal Medicine 17 Ed. 2008].

General symptoms Diabetes mellitus

- Increased thirst
- Frequent urination, passing large quantities of urine, hence dehydration
- Increased hunger
- Feeling very tired without any particular reason
- Blurred vision due to dehydration of eye lens
- Continuous ache, pain in legs and feet including numbness, burning sensation, or no sensation
- No healing of cuts, wounds, boils and sores
- Skin infection, especially around genital area, vaginal infection in women, urinary tract infection
- Impotence
- Weight loss

Types of Diabetes

Diabetes basically can be categorized into two types (WHO, 1985). Diabetes insipides and Diabetes mellitus.

Types of Diabetes mellitus

There are several types of diabetes mellitus. The following classification system for diabetes was endoned by the board of directors of the American Diabetes Association at its 1979 annual meeting and also by the World Health Organization (1994).

I. Type 1 diabetes (-cell destruction, usually leading to absolute insulin deficiency)

A. Immune-mediated
B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretary defect with insulin resistance)

Other specific types of diabetes

A. Genetic defects of -cell function characterized by mutations in:

- Hepatocyte nuclear transcription factor (HNF) 4 (MODY 1)

- Glucokinase (MODY 2)
- HNF-1 (MODY 3)
- Insulin promoter factor (IPF) 1 (MODY 4)
- HNF-1 (MODY 5)
- Mitochondrial DNA
- Proinsulin or insulin conversion

B. Genetic defects in insulin action

- Type A insulin resistance
- Leprechaunism
- Rabson-Mendenhall syndrome
- Lipoatrophic diabetes

C. Diseases of the exocrine pancreas-pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculous pancreatopathy

D. Endocrinopathies acromegaly, Cushing's syndrome, glucagonoma

pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma

E. Drug- or chemical-induced Vacor, pentamidine, nicotinic acid, glucocorticoids, thyroid hormone, diazoxide, -adrenergic agonists, thiazides, phenytoin, interferon, protease inhibitors, clozapine, beta blockers

F. Infections: congenital rubella, cytomegalovirus, coxsackie

G. Uncommon forms of immune-mediated diabetes:

"stiff-man" syndrome, anti-insulin receptor antibodies.

H. Other genetic syndromes sometimes associated with diabetes:

Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Wolfram's syndrome, Friedreich's ataxia, Huntington's chorea, Laurence-Moon-Biedl syndrome, myotonic dystrophy, porphyria, Prader-Willi syndrome

I. Gestational diabetes mellitus (GDM)

J. MODY: Maturity onset of diabetes of the young.

TYPE-I DIABETES MELLITUS

(INSULIN DEPENDENT DIABETES MELLITUS)

Genetics

The genetic contributions to type 1 DM involve multiple genes. The development of the disease appears to require inheritance of a sufficient complement of genes to confer susceptibility to the disorder. The concordance of type 1 DM in identical twins ranges between 30 and 70%, indicating that additional modifying factors must be involved in determining whether diabetes develops.

The major susceptibility gene for type 1 DM is located in the HLA region on chromosome 6. Polymorphisms in the HLA complex appear to account for 40 to 50% of the genetic risk of developing type 1 DM.

Most individuals with type 1 DM have the HLA DR3 and/or DR4 haplotype. Genes that confer protection against the development of the disease also exist. For example, the haplotype DQA1*0102, DQB1*0602 is present in 20% of the U.S. population but is extremely rare in individuals with type 1DM (<1%).

Environmental Factors

It has been proposed that lack of exposure to pathogenic organisms in early childhood limits maturation of the immune system and increases susceptibility to autoimmune disease (‘the hygiene hypothesis’), Viruses, Diet, Stress, Immunological factors

Viruses

The evidence that viral infection might cause some forms of type 1 diabetes is derived from studies where virus particles known to cause cytopathic or autoimmune damage to beta cells. The viruses have been isolated from the pancreas.Viruses that causes type 1 diabetes include Mumps, Coxsackie B4, retroviruses, rubella (in utero), cytomegalovirus and Epstein-Barr virus.

Diet

Dietary factors may influence the development of type 1 diabetes. Bovine serum albumin (BSA), a major constituent of cow's milk, has been implicated in triggering type 1 diabetes. It has been shown that children who are given cow's milk early in infancy are more likely to develop type 1 diabetes than those who are breastfed. BSA may cross the neonatal gut and raise antibodies which, because of the close homology between BSA, the Beta chain of HLA class II antigens and a heat-shock protein expressed by beta cells, could cross-react with and cause damage to beta cell components.Various nitrosamines and coffee have been proposed as potentially diabetogenic factors.

Stress

Stress may progress the development of type 1 diabetes by stimulating the secretion of counter-regulatory hormones and possibly by modulating immune activity.

Immunological Factors

Type 1 diabetes is a slow T cell-mediated autoimmune disease. Family studies have produced evidence that destruction of the insulin-secreting cells in the pancreatic islets takes place over many years. Hyperglycaemia accompanied by the classical symptoms of diabetes occurs only when 70-90% of beta cells have been destroyed. In humans and animals with spontaneous type 1 diabetes the immune system retains the capacity to recognize and destroy transplanted pancreatic beta cells indefinitely.

TYPE-II DIABETES MELLITUS

(NON INSULIN DEPENDENT DIABETES MELLITUS)

Type 2 diabetes commonly occurs in subjects who are obese and insulin-resistant, but these two factors alone are insufficient to cause diabetes unless accompanied by impaired beta cell function.

Genetics

Type 2 DM has a strong genetic component. Although the major genes that predispose to this disorder have yet to be identified, it is clear that the disease is polygenic and multifactorial.

Various genetic loci contribute to susceptibility, and environmental factors (such as nutrition and physical activity) further modulate phenotypic expression of the disease. The concordance of type 2 DM in identical twins is between 70 and 90%. Individuals with a parent with type 2 DM have an increased risk of diabetes; if both parents have type 2 DM, the risk in offspring may reach 40%. Insulin resistance, as demonstrated by reduced glucose utilization in skeletal muscle, is present in many nondiabetic, first-degree relatives of individuals with type 2 DM. However, definition of the genetic abnormalities of type 2 DM remains a challenge because the genetic defect in insulin secretion or action may not manifest itself unless an environmental event or another genetic defect, such as obesity, is superimposed.

Environmental Factors

1. Life Style

Epidemiological studies of type 2 diabetes provide evidence that over eating, especially when combined with obesity, middle-aged people with diabetes eat significantly more and are fatter and less active than their non-diabetic siblings. Obesity probably acts as a diabetogenic factor (through increasing resistance to the action of insulin) in those genetically predisposed to develop type 2 diabetes.

2. Malnutrition in utero

Retrospective analysis of the birth weight of males has born an inverse relationship between weight at birth and at 1 year, and the development of type 2 diabetes in late adulthood. It is proposed (but not yet proven) that malnutrition in utero may programme beta cell development and metabolic functions at a critical period, so predisposing to type 2 diabetes later in life. Smoking during pregnancy has also been implicated.

3. Age

Age is an important risk factor for type 2 diabetes. Over 70% of all cases of diabetes occur after the age of 50 years. Type 2 diabetes is principally a disease of the middle aged and elderly, affecting 10% of the population over the age of 65.

4. Pregnancy

During normal pregnancy, insulin sensitivity is reduced through the action of placental hormones and this affects glucose tolerance. The term 'gestational diabetes' refers to hyperglycaemia occuring for the first time during pregnancy. Repeated pregnancy may increase the likelihood of developing irreversible diabetes, particularly in obese women; 80% of women with gestational diabetes ultimately develop permanent clinical diabetes requiring treatment.

Pathogenesis of type 2 diabetes

Insulin resistance

Increased hepatic production of glucose and resistance to the action of insulin in muscle are invariable in both obese and non-obese patients with type2 diabetes. Insulin resistance may be due to any one of three general causes: an abnormal insulin molecule, an excessive amount of circulating antagonists, or target tissue defects. The last is the most common cause of insulin resistance in type 2 diabetes and seems to be the predominant abnormality in those with more severe hyperglycaemia.

A characteristic feature of type 2 diabetes is that it is often associated with other medical disorders including obesity, hypertension and hyperlipidaemia. It has been suggested that this cluster of conditions, all of which predispose to cardiovascular disease, is a specific entity (the 'insulin resistance syndrome' or 'metabolic syndrome'), with insulin resistance being the primary defect.

Pancreatic beta cell failure

In type 2 diabetes there is only moderate reduction in the total mass of pancreatic islet tissue which is consistent with a measurable fall in plasma insulin concentration when related to the blood glucose level. However, some pathological

changes are typical of type 2 diabetes, the most consistent of which is deposition of amyloid. This is accompanied by atrophy of the normal tissue, particularly islet epithelial cells. Islet amyloid is composed of insoluble fibrils formed from islet amyloid polypeptide (also known as amylin). Small quantities of islet amyloid are very common in elderly non-diabetic patients, and the role of islet amyloid in the pathogenesis of type 2 diabetes is uncertain. Deposition of amyloid is probably not a cause of diabetes but rather reflects a pathological process which is increased in type 2 diabetes. More extensive amyloidosis is, however, found in patients who have progressed to insulin replacement therapy, suggesting that islet function may become compromised by amyloid deposition.

While beta cell numbers are reduced by 20-30% in type 2 diabetes, alpha cell mass is unchanged and glucagons secretion is increased, which may contribute to the hyperglycaemia. Insulin resistance tends to raise blood glucose and this stimulates insulin secretion to prevent hyperglycaemia . when the maximal insulin secretory capacity has been exceeded, any further increase in fasting blood glucose levels causes a decline in insulin generation. Possible mechanisms for beta cell decompensation include glucotoxicity, an intrinsic failure of insulin production, a switch to abnormal processing pathways producing biologically inactive products and chronic degranulation of the beta cell.

GESTATIONAL DIABETES

Gestational diabetes, defined as hyperglycaemia diagnosed for the first time in pregnancy, is a common problem. It occurs in individuals who have an inherited predisposition to develop diabetes and may take the form of either type I or type II diabetes. The hyperglycaemia may not disappear after delivery. It is associated not only with increased rates of perinatal mortality and neonatal morbidity but also with a high incidence (possibly as great as 80% at 25 years postpartum) of subsequent clinical diabetes (both type I and type II) in the mother. Normalisation of metabolism, whether by treatment with dietary measures alone or, more commonly, with additional treatment in the form of insulin, undoubtedly reduces the fetal risk; its effect on diminishing the maternal risk of subsequent diabetes is less certain.

MAJOR MANIFESTATIONS OF DISEASE

Hyper Glycaemia:

Hyperglycaemia is a very common biochemical abnormality. It is frequently detected on routine biochemical analysis of asymptomatic patients, and is found during conditions which impose a burden on pancreatic beta cells, such as pregnancy, severe illness or treatment with drugs such as corticosteriods('stress hyperglycaemia').

Symptoms of Hyperglycemia Associated With Diabetes

- Thirst, dry mouth.
- Poly uria.
- Nocturia.
- Tiredness, fatigue, irritability.
- Recent change in weight.
- Blurring of vision.
- Pruritus vulvae, balanitis(genital condidiasis)
- Nausea; headache
- Hyperphagia; predilection for sweet foods

Diabetic Ketoacidosis:

Keto acidosis is caused by insulin deficiency and an increase in catabolic hormones, leading to hepatic over-production of glucose and ketone bodies. The cardinal biochemical features of diabetic ketoacidosis are

- Hyperglycaemia
- Hyperketonaemia
- Metabolic acidosis

COMPLICATIONS OF DIABETES MELLITUS

1. Acute Complications

- Diabetic ketoacidosis (DKA)
- Nonketotic hyperosmolar state(NKHS)

Diabetic Ketoacidosis:

Diabetic ketoacidosis is a major medical emergency and remains a serious cause of morbidity, principally in people with type 1 diabetes. The average mortality in developed countries is 5-10% and is higher in the elderly.

A clear understanding of the biochemical basis and pathophysiology of this problem is essential for its efficient treatment.

Ketoacidosis is caused by insulin deficiency and an increase in catabolic hormones, leading to hepatic over-production of glucose and ketone bodies.

The cardinal biochemical features of diabetic ketoacidosis are:

- hyperglycaemia
- hyperketonaemia
- metabolic acidosis.

Hyperglycaemia causes a profound osmotic diuresis leading to dehydration and electrolyte loss, particularly of sodium and potassium. The metabolic acidosis forces hydrogen ions into cells, displacing potassium ions, which may be lost in urine or through vomiting.

About half the deficit of total body water is derived from the intracellular compartment and occurs comparatively early in the development of acidosis with relatively few clinical features; the remainder represents loss of extra cellular fluid sustained largely in the later stages. It is at this time that marked contraction of the size of the extra cellular space occurs, with haemoconcentration, a decreased blood volume, and finally a fall in blood pressure with associated renal ischaemia and oliguria.

Every patient in diabetic ketoacidosis is potassium-depleted, but the plasma concentration of potassium gives very little indication of the total body deficit. Plasma potassium may even be raised initially due to disproportionate loss of water and catabolism of protein and glycogen.

However, soon after insulin treatment is started there is likely to be precipitous fall in the plasma potassium due to dilution of extra cellular potassium by administration

of intravenous fluids, the movement of potassium into cells as a result of treatment with insulin, and the continuing renal loss of potassium.

The severity of ketoacidosis can be assessed rapidly by measuring the plasma bicarbonate; less than 12 mmol/l indicates severe acidosis.

Clinical features:

a.Symptoms - Nausea,vomitingThirst,polyuria,Abdominal pain,Altered mental function and Shortness of breath

b.Physical findings - Tachycardia,Dry mucous membranes, reduced skin turgor,Dehydration, hypotension,Tachypnea, Kussmaul respirations, respiratory distress,Abdominal tenderness, (may resemble acute pancreatitis or surgical abdomen)FeverLethargy ,obtundation , cerebral edema and possibly comac.

c.Precipitating events - Inadequate insulin administration, infection (pneumonia/ UTI/ gastroenteritis / sepsis), Infarction (cerebral, coronary, mesenteric, peripheral) and drugs (cocaine)

Nonketotic Hyperosmolar State

Clinical Features - NKHS is most commonly seen in elderly individuals with type 2 DM. Its most prominent features include polyuria; orthostatic hypotension; and a variety of neurologic symptoms that include altered mental status, lethargy, obtundation, seizure, and possibly coma.

The prototypical patient is a mildly diabetic, elderly individual with a several week history of polyuria, weight loss, and diminished oral intake that culminates in mental confusion, lethargy, or coma. The physical examination reflects profound dehydration and hyperosmolality and reveals hypotension, tachycardia, and altered mental status

NKHS is often precipitated by a serious, concurrent illness such as myocardial infarction or stroke. Sepsis, pneumonia, and other serious infections are frequent precipitants and should be sought thoroughly. In addition, a debilitating condition (prior stroke or dementia) or social situation that compromises water intake may contribute to

the development of the disorder. Finally, the development of NKHS can be associated with the use of certain medications (thiazide diuretics, glucocorticoids, phenytoin).

2. Chronic Complications of Diabetes Mellitus

a. Microvascular

- Eye disease
- Retinopathy(nonproliferative/proliferative)
- Macular edema
- Cataracts
- Glaucoma
- Neuropathy
- Sensory and motor (mono- and polyneuropathy)
- Nephropathy.

b. Macrovascular

- Coronary artery disease
- Peripheral vascular disease
- Cerebrovascular disease

c. Other

- Gastrointestinal (gastroparesis, diarrhea)
- Genitourinary (uropathy/sexual dysfunction)
- Dermatologic

DIABETIC RETINOPATHY

Diabetic retinopathy is the most common cause of blindness in adults between 30 and 65 years of age in developed countries.

Pathogenesis

Hyperglycaemia increases retinal blood flow and metabolism and has direct effects on retinal endothelial cells and pericytes, loss of which impairs vascular autoregulation.

The resulting uncontrolled blood flow increases production of vasoactive substances and endothelial cell proliferation, resulting in capillary closure. This causes chronic retinal hypoxia and stimulated production of growth factors, including vascular endothelial growth factor (VEGF). VEGF acts via protein kinase C to stimulate endothelial cell growth (causing new vessel formation) and increased vascular permeability (causing exudative damage).

Clinical Features Of Diabetic Retinopathy

- Microaneurysms
- Retinal haemorrhages
- Exudates
- Cotton wool spots
- Venous changes
- No vascularisation
- Pre-retinal haemorrhage
- Vitreous haemorrhage
- Fibrosis

Microaneurysms

In most cases these are the earliest clinical abnormality detected. They appear as tiny, discrete, circular, dark red spots near to, but apparently separate from, the retinal vessels. They look like tiny haemorrhages but photographs of injected preparations of retina show that they are in fact minute aneurysms arising mainly from the venous end of capillaries near areas of capillary closure.

Haemorrhages

These most characteristically occur in the deeper layers of the retina and hence are round and regular in shape and described as ‘blot’ haemorrhages. The smaller ones may be difficult to differentiate from microaneurysms and the two are often grouped together as ‘dots and blots’. Superficial flame-shaped haemorrhages may also occur, particularly if the patient is hypertensive.

Exudates

These are characteristic of diabetic retinopathy. They vary in size from tiny specks to large confluent patches and tend to occur particularly in the perimacular area. They result from leakage of plasma from abnormal retinal capillaries and overlie areas of neuronal degeneration.

Cotton Wool Spots

These are similar to those seen in hypertension, and also occur particularly within five disc diameters of the optic disc. They represent arteriolar occlusions causing retinal ischaemia and hence are a feature of pre-proliferative diabetic retinopathy; they are most often seen in rapidly advancing retinopathy or in association with uncontrolled hypertension.

Intraretinal microvascular abnormalities

Intraretinal microvascular abnormalities (IRMA) are dilated, tortuous capillaries which represent the remaining patent capillaries in an area where most have been occluded.

Neovascularisation

This may arise from the venous circulation on the optic disc or the retina in response to areas of ischaemic retina. The earliest appearance is that of fine tufts of delicate vessels forming arcades on the surface of the retina. As they grow, they may extend forwards towards the vitreous.

This first appears as a white, cloudy haze among the network of new vessels. As it extends, the new vessels may be obliterated and the surrounding retina covered by a dense white sheet. At this stage, bleeding is less common but retinal detachment can occur due to contraction of adhesions between the vitreous and the retina.

Venous Changes

These include venous dilatation (an early feature probably representing increased blood flow), ‘beading’ (sausage-like changes in calibre) and increased tortuosity including ‘oxbow lakes’ or loops.

These latter changes indicate widespread capillary non-perfusion and are a feature of advanced pre-proliferative retinopathy.

Cataract

Cataract is permanent lens opacity and is the most common cause of visual deterioration in the elderly population.

The lens thickens and pacifies with age, and the increased metabolic insult to the lens in people with diabetes causes these changes to accelerate and occur prematurely. Very rarely, a type of cataract specific to diabetes occurs in young patients with poorly controlled diabetes, called a 'snow-flake' cataract. This does not usually affect vision but tens to make fundal examination difficult.

Renal Complications of Diabetes Mellitus

Diabetic nephropathy is the leading cause of ESRD in the United States and a leading cause of DM-related morbidity and mortality. Proteinuria in individuals with DM is associated with markedly reduced survival and increased risk of cardiovascular disease. Individuals with diabetic nephropathy almost always have diabetic retinopathy also.

Like other microvascular complications, the pathogenesis of diabetic nephropathy is related to chronic hyperglycemia. The mechanisms by which chronic hyperglycemia leads to ESRD, though incompletely defined, involve the following: interaction of soluble factors (growth factors, angiotensin II, endothelin, AGEs), hemodynamic alterations in the renal microcirculation (glomerular hyperfiltration, increased glomerular capillary pressure), and structural changes in the glomerulus (increased extracellular matrix, basement membrane thickening, mesangial expansion, fibrosis). Some of these effects may be mediated through angiotensin receptors. Smoking accelerates the decline in renal function.

The natural history of diabetic nephropathy is shown schematically in and is characterized by a fairly predictable pattern of events. Although this sequence of events was defined for individuals with type 1 DM, a similar pattern is also likely in type 2 DM. Glomerular hyperfusion and renal hypertrophy occur in the first years after the onset of DM and are reflected by an increased glomerular filtration rate (GFR).

During the first 5 years of DM, thickening of the glomerular basement membrane, glomerular hypertrophy, and mesangial volume expansion occur as the GFR returns to normal. After 5 to 10 years of type 1 DM, ~40% of individuals begin to excrete small amounts of albumin in the urine (microalbuminuria). Microalbuminuria is defined as 30 to 300 mg/d in a 24-h collection or 30 to 300 ug/mg creatinine in a spot collection.

The appearance of microalbuminuria (incipient nephropathy) in type 1 DM is a very important predictor of progression to overt proteinuria (>300 mg/d). Blood pressure may rise slightly at this point but usually remains in the normal range. Once overt proteinuria is present, there is a steady decline in GFR, and ~50% of individuals reach ESRD in 7 to 10 years. The early pathologic changes and albumin excretion abnormalities are reversible with normalization of plasma glucose. However, once nephropathy becomes overt, the pathologic changes are likely irreversible.

The nephropathy that develops in type 2 DM differs from that of type 1 DM in the following respects:

- Microalbuminuria or over nephropathy may be present when type 2 DM is diagnosed, reflecting its long asymptomatic period;
- Hypertension more commonly accompanies microalbuminuria or overt nephropathy in type 2 DM; and
- Microalbuminuria may be less predictive of progression to overt nephropathy in type 2 DM. Finally, it should be noted that albuminuria in type 2 DM may be secondary to factors unrelated to DM, such as hypertension, congestive heart failure, prostate disease, or infection.

Other renal problems may also occur in individuals with DM. Type IV renal tubular acidosis (hyporeninemic hypoaldosteronism) occurs in many individuals with DM.

NEUROPATHY AND DIABETES MELLITUS

Diabetic neuropathy occurs in approximately 50% of individuals with long-standing type 1 and type 2 DM. It may manifest as polyneuropathy, mononeuropathy, and/or autonomic neuropathy.

Polyneuropathy / Mononeuropathy: The most common form of diabetic neuropathy is distal symmetric polyneuropathy. It most frequently presents with distal sensory loss.

Hyperesthesia, parathesia, and pain also occur. Any combination of these symptoms may develop as neuropathy progresses. Physical examination reveals sensory loss, loss of ankle reflexes, and abnormal position sense.

Diabetic polyradiculopathy is a syndrome characterized by severe disabling pain in the distribution of one or more nerve roots. It may be accompanied by motor weakness. Intercostal or truncal radiculopathy causes pain over the thorax or abdomen. Involvement of the lumbar plexus or femoral nerve may cause pain in the thigh or hip and may be associated with muscle weakness in the hip flexors or extensors (diabetic amyotrophy). Fortunately, diabetic polyradiculopathies are usually self-limited and resolve over 6 to 12 months.

Mononeuropathy (dysfunction of isolated cranial or peripheral nerves) is less common than polyneuropathy in DM and presents with pain and motor weakness in the distribution of a single nerve. A vascular etiology is favored, but the pathogenesis is unknown. Involvement of the third cranial nerve is most common and is heralded by diplopia.

Autonomic Neuropathy Individuals with long-standing type 1 or 2 DM may develop signs of autonomic dysfunction involving the cholinergic, noradrenergic, and peptidergic (peptides such as pancreatic polypeptide, substance P, etc.) systems. DM-related autonomic neuropathy can involve multiple systems including: the cardiovascular, gastrointestinal, genitourinary, sudomotor, and metabolic systems.

Autonomic neuropathies affecting the cardiovascular system cause a resting tachycardia and orthostatic hypotension. Reports of sudden death have also been attributed to autonomic neuropathy. Gastroparesis and bladder-emptying abnormalities are also likely related to the autonomic neuropathy seen in DM (discussed below). Hyperhidrosis of the upper extremities and anhidrosis of the lower extremities result from sympathetic nervous system dysfunction. Anhidrosis of the feet can promote dry skin with cracking, which increases the risk of skin ulceration.

Autonomic neuropathy may reduce counterregulatory hormone release, leading to an inability to sense hypoglycemia appropriately hypoglycemia unawareness; thereby subjecting the patient to the risk of severe hypoglycemia and complicating efforts to improve glycemic control.

Cardiovascular Morbidity and Mortality

Cardiovascular disease is increased in individuals with type 1 or type 2 DM. The Framingham Heart Study revealed a marked increase in several cardiovascular diseases in DM including peripheral vascular disease, congestive heart failure, coronary artery disease, myocardial infarction, and sudden death (risk increase from one- to fivefold).

The extremely high frequency of underlying cardiovascular disease in individuals with diabetes (especially in type 2 DM).The absence of chest pain ("silent ischemia") is common in individuals with diabetes.

Cardiovascular Risk Factors

Dyslipidemia Individuals with DM may have several forms of dyslipidemia. Because of the additive cardiovascular risk of hyperglycemia and hyperlipidemia, lipid abnormalities should be aggressively detected and treated as part of comprehensive diabetes care. The most common pattern of dyslipidemia is hypertriglyceridemia and reduced HDL cholesterol levels. DM itself does not increase levels of LDL, but the small dense LDL particles found in type 2 DM are more atherogenic because they are more easily glycated and susceptible to oxidation.

Hypertension: Hypertension can accelerate other complications of DM, particularly cardiovascular disease and nephropathy. Hypertension therapy should first emphasize life-style modifications such as weight loss, exercise, stress management, and sodium restriction.

DIABETIC FOOT

The foot is a frequent site for complication in patients with diabetes and for this reason foot care is particularly important. Tissue necrosis in the feet is a common reason for hospital admission in diabetic patient. Such admission tend to be prolonged and often end with amputation. The clinical features are listed below

Symptoms

- Paresthesia
- Pain
- Numbness

Structural Damage

- Ulcer
- Sepsis
- Abscess
- Osteomyelitis
- Digital gangrene
- Charcot joint

Management of Diabetic Foot Ulcers

Remove callus skin, treat infection, avoid weight-bearing, ensure good diabetic control, control oedema, undertake angiogram to assess feasibility of vascular reconstruction where indicated.

Clinical examination of the patient with diabetes

1. **Examination Of The Hands**
   - Limited joint mobility (sometimes called cheirorthropathy) may be present; this is the inability to extend (to 180) the metacarpophalangeal or interphalangeal joints of at least one finger bilaterally. The effect can be demonstrated in the prayer sign. It causes painless stiffness in the hands, and occasionally affects the wrists and shoulders.
   - Dupuytren's contracture is common in diabetes and may include nodules or thickening of the skin and knuckle pads.
   - Carpal tunnel syndrome is common in diabetes and presents with wrist pain radiating into the hand.
   - Trigger finger (flexor tenosynovitis) may be present in people with diabetes.
   - Muscle-wasting/sensory changes may be present as features of a peripheral sensorimotor neuropathy, although this is more common in the lower limbs.

2.Abdomen

Hepatomegaly

3.Blood Pressure

4.Axilla

Acanthosis nigricans

5.Neck

Carotid pulses, Bruits and thyroid enlargement

6.Head

Xanthelasma, Cranial nerve palsy and eye movements/ptosis

7.Examination Of The Eyes

- Visual acuity
- Distance vision using Snellen's chart at 6 metres.
- Near vision using standard reading chart.

Impaired visual acuity may indicate the presence of diabetic eye disease, and serial decline may suggest development or progression in severity. Lens opacification, look for the red reflex using the ophthalmoscope held 30 cm from the eye. The presence of lens opacities or cataract should be noted.

Fundus examination

The pupils must be dilated with a mydriatic and examined in a darkened room. Features of diabetic retinopathy should be noted, including evidence of previous laser treatment which leaves photocoagulation scars.

8.Insulin Injection Sites

Main areas used are

- Anterior abdominal wall
- Upper thighs/buttocks
- Upper outer arms

Inspection

- Bruising
- Lumps (lipodystrophy)

- Subcutaneous fat loss (lipoatrophy; associated with injection of unpurified animal insulins-now rare)
- Erythema, infection (rare)

9.Legs

- Muscle-wasting
- Sensory abnormality
- Granuloma annulare
- Hair loss
- Tendon reflexes
- Necrobiosis lipoidica
- Neuropathic foot ulcer

10.Examination Of The Feet

Inspection

Look for evidence of callus formation on weight-bearing areas, clawing of the toes (a feature of neuropathy, loss of the plantar arch, discoloration of the skin,ischaemia), localised infection and the presence of ulcers. Deformity of the feet may be present, especially in charcot neuroarthropathy.Fungal infection may affect skin between toes, and nails.

Circulation

Peripheral pulses, skin temperature and capillary refill should be tested.

Sensation

- **Light touch**: use monofilaments.
- **Vibration sense**: use 128Hz tuning fork over big toe/malleoli.
- **Pin-prick**: Use pin
- **Pain**: pressure over Achilles tendon.
- **Proprioception test:**

  Position of big toe

  Test for distal anaesthesia/hyperaesthesia in stocking distribution.

Reflexes

Test plantar and ankle reflexes

Management

Aims of treatment

- Adequate control of hyperglycemia and glycosuria
- Prevention of complications
- Disappearance of diabetic symptoms
- Maintenance the appropriate body weight

All of these, the first two are very important.

Treatments

There are 4 methods of treatments available for diabetic patients

- Ladder diet regime
- Diet and oral hypoglycemic agents
- Diet and insulin
- Special treatment for complications

DIETARY MANAGEMENT

Aims of Dietary Management

- Abolish symptoms of hyperglycaemia
- Reduce overall blood glucose and minimise fluctuations
- Achieve weight reduction in obese patients to reduce insulin resistance, hyperglycaemia and dyslipidaemia
- Avoid hypoglycaemia associated with therapeutic agents (insulin, sulphonylureas)
- Avoid weight gain associated with therapeutic agents (insulin, sulphonylureas, thiazolidinediones)
- Avoid 'atherogenic' diets or those which may aggravate diabetic complications (e.g. high protein intake in nephropathy)

General Principles of Diet for Diabetes:

Direction sugar intake in the form of refined carbohydrates should be totally avoided. This includes table sugar, sweets, and jaggery. The total quantity of food must be restricted.

There is no need to change over from rice to wheat or ragi as the carbohydrate content of these different cereals is not significantly different. Green leafy vegetables and other low calorie foods can be taken in unlimited quantities.

Addition of vegetable proteins in the form of bengal gram, green gram, have multiple benefits as they:

a).Increase the protein content

b).Increase the fibre content

c).Help to flatten sudden urges of blood sugar after a meal

d).Help to reduce serum lipid (fat) levels

e).The diet should help to maintain ideal body weight.

f).The diet should also help bring down the cholesterol triglyceride levels

Types of Diabetic Diet

The basis types of diet are used in the treatment of diabetes;

1. Low energy, weight-reducing diets
2. Weight maintenance diets
3. Diets for insulin-treated diabetes

Low-energy, weight-reducing diets

Dietary prescriptions which cause a daily deficit of 500 kcal provide a realistic diet and induce a weekly weight loss of around 0.5 kg. Rapid weight reduction may provoke loss of lean body tissue, and care must be taken in the elderly to avoid the omission of essential nutrients, vitamins and minerals. Caloric restriction is essential for the obese diabetic patient treated with insulin and most oral agents, to try to minimise the weight

gain which these can promote. In such individuals, the omission of snacks between meals is often necessary.

Weight maintenance diets

These are necessary for individuals with a normal body mass index and ideally should be high in carbohydrate and low in fat, with particular attention being paid to the type of fat ingested.

Diets for Insulin-Treated Diabetes

A regular pattern of meals (and snacks) is important to maintain a constant daily intake of carbohydrate, and protects against hypoglycaemia. Simple information on the relative carbohydrate content of foods can be provided where appropriate. Carbohydrate exchanges (10 g portions) are currently not advocated as a method of controlling carbohydrate intake, as the exchange system makes no allowance for the glycaemic effect or for the fat content of foods. However, a good working knowledge of the carbohydrate content of foods is essential for practical management. An insufficient dose of insulin for a meal with a large carbohydrate content leads to post-prandial hyperglycaemia, while inadequate carbohydrate consumption risks hypoglycaemia.

Diabetic Foods and Sweeteners

Low-calorie and sugar-free drinks are useful for patients with diabetes. These drinks usually contain non-nutritive sweeteners. Many 'diabetic foods' contain sorbitol or fructose which are relatively high in energy, may be expensive and may have gastrointestinal side-effects. They are not recommended as part of the diabetic diet.

The non-nutritive sweeteners saccharin, aspartame, sucramate and acesulphame K are the most widely used and provide means for reducing energy intake without loss of palatability.

TRIAL DRUG

ATTHIPPATTAIYATHI KASAYAM

“அத்திமரப் பட்டை நன்னாரி ஆவாரை கடலிறஞ்சி
யித்துடன் மருதிலுப்பை இசைந்தநீர்ப் பூலாக்கொன்றை
உத்ததோர் புளியம்பட்டை சிறுகீரை சீந்தில்முத்த
நத்தைச்சூரி வேர்கற்றாழை நாரத்தை வேருடங்கூட்டே

கொள்ளாய் தாளிசபத்திரி திரிகடுகு குலவுபலங்க ளிரண்டாக
வில்ளாய் சாதிப்பத்திரி காயம் மிகுத்த கிராம்பு வராகனெடை
மெள்ளாய் சூரண மாய்ப்பண்ணி வெருகடிநாழி நீருழக்காய்க்
கொள்ளாய் கசாய மிருபதுநாள் கூட்டாயெருமை மோர்தயிர்”

-அகத்தியர் இரண்டாயிரம்.

INGREDIDENTS:

Required raw drugs:

1.	Ficus recemosa,Linn	-*Atthi*	- bark -70gm
2.	Cassia fistula,Linn	-*Kontrai*	- bark -70gm
3.	Cassia auriculata,Linn	-*Aavaram*	- bark -70gm
4.	Salacia reticulate,Wight	-*Kadalalinjil*	- bark -70gm
5.	Madhuca longifolia (Koenig) J. F. Macbr	-*Iluppai*	- bark -70gm
6.	Tamarindus indica,Linn	-*Puli*	- bark -70gm
7.	Terminalia arjuna (Roxb)	-*Maruthu*	- bark -70gm
8.	Spermacoce hispida,Linn	-*Nathaisoori*	- root -70gm
9.	Citrus medica,Linn	-*Naarathai*	- root -70gm
10.	Hemidesmus indicus,Linn R.Br	-*Nannaari*	- root -70gm
11.	Amaranthus tricolor,Linn	-*Sirukeerai*	- root -70gm
12.	Phyllanthus reticulates,Poir	-*Neerpoola*	- root -70gm
13.	Aloe barbadensis,Miller	-*Kattralai*	- root -70gm
14.	Cyperus rotundus,Linn	-*Korai*	- root -70gm
15.	Tinospora cordifolia (Willd)	-*Seenthil*	-stem -70gm
16.	Zingiber officinale,Rosc	-*Sukku*	-rhizome-70gm
17.	Piper nigrum,Linn	-*Milaku*	-fruit -70gm
18.	Piper longum,Linn	-*Thippili*	-fruit -70gm
19.	Abies spectabilis (D.Don) Mirb	-*Thalisa pathri*	-leaf -70gm
20.	Myristica fragrans,Houtt	-*Saathi pathri*	- aril -4.2gm
21.	Syzygium aromaticum,(Linn) Merrill & Perry	-*Kirambu*	-bud -4.2gm
22.	Ferula asafoetida, Linn	-*Perungaayam*	-gum resin-4.2gm

PROPERTIES OF THE TRIAL DRUGS

1.அத்திப்பட்டை

வீறு கடுப்பிரத்தம் வெண்சீத ரத்தமொடு
நாறுவிர ணங்களெலாம் நாடாவாம்- கூறுங்கால்
அத்திதரு மேகம்போம் ஆயிழையே! எஞ்ஞான்றும்
அத்திப்பாற் பட்டைக் கறி.

-அ.கு.

Botanical Name	**:**	Ficus racemosa, Linn
Family	**:**	Moraceae
Suvai	**:**	Thuvarppu
Thanmai	**:**	Thatpam
Pirivu	**:**	Enippu
Part Used	**:**	Bark
Properties	**:**	Astringent

2.ஆவாரை பட்டை

சொல்லுதற்கு மட்டோ தொலையதா மேகநீர்
எல்லா மெழிக்கு மெரிவகற்று- மெல்லவச
மாவாரைப் பம்பரம்போ லாட்டுந் தொழிலணங்கே!
யவாரை மூலி யது.

-அ.கு.

Botanical Name	**:**	Cassia auriculata, Linn
Family	**:**	Caesalpiniaceae
Suvai	**:**	Thuvarppu
Thanmai	**:**	Thatpam
Pirivu	**:**	Enippu
Part Used	**:**	Bark
Properties	**:**	Astringent, Tonic

3.இலுப்பைப்பட்டை

புண்ணும் புரையுமறும் போதத் துவர்ப்பாகும்
எண்ணுமகக்கடுப்பி ருக்குமோ- பெண்ணே கேள்
நீரிழிவு மேகும் நெடுமோமை மூலத்தாள்
போரடர்க டுப்பிரத்தம் போம்.

மந்த மரோசிம காசூலை சர்வசுரம்
விந்துநட்டந் தாகமொடு மெய்யிளைப்பும்- முந்த
அலுப்பைப்பெற் றேகு மளகத்திற் காகா
இலுப்பைக்குச் சூடதிக மெண்.

-அ.கு.

Botanical Name	**:**	Madhuca longifolia (Koenig) J. F. Macbr
Family	**:**	Sapotaceae
Suvai	**:**	Thuvarppuy
Thanmai	**:**	Thatpam
Pirivu	**:**	Karppu
Part Used	**:**	Bark
Properties	**:**	Alterative, stimulant, astringent, tonic, stomachic

4.கடலழிஞ்சில் பட்டை

தீதில் உடலழிஞ்சில் செய்யுங் குணங்கேளாய்
ஓதுமது மேக மொழிப்பதல்லால்- வதத்தில்
வந்தசலம் பித்தசல மாகபச்ச லந்தாகத்
தொந்தசல மும்போக்குஞ் சொல்.

கமல விரணங் கசிநீ ரகற்றும்
நிமல சுரம்விலக்கு நேரே- கமலமு
மாதே! கடலழிஞ்சில் வன்பட்டை பேதிகட்டுஞ்
சீதாமே கம்போக்குந் தேர்.

-அ.கு.

Botanical Name	**:**	Salacia oblonga, wall
Family	**:**	Celastraceae
Suvai	**:**	Thuvarppu
Thanmai	**:**	Thatpam
Pirivu	**:**	Karppu
Part Used	**:**	Root bark
Properties	**:**	Demulcent, astringent

5.கொன்றைப் பட்டை

பாண்டரங்கர் பூணாய்ப் பறக்கடித்து மேகத்தை
யாண்டாங்கக் கைக்குள்வச மாக்குமே- காண்டற்
குதவிசில செய்துடலை யோம்புமிது நீபார்
இதழியெனுங் கொன்றைபுவி யில்.

-தே.வெண்பா.

குட்டங் கிருமி கொடுஞ்சூலை வாதமையம்
துட்ட மலமருசி தூரப்போம்- தட்டிச்
சுரக்கின்ற பேதியுண்டாம் துய்க்கத் துவர்கும்
சரக்கொன்றைக் காரணங்கே! சாற்று.

-அ.கு.

Botanical Name	:	Cassia fistula, Linn
Family	:	Caesalpiniaceae
Suvai	:	Kaippu, thuvarppu
Thanmai	:	Veppam
Pirivu	:	Karppu
Part Used	:	Bark
Properties	:	Laxative, astringent.

6.மருதம் பட்டை

ஓதமெனு நீரிழிவை யோட்டும் பிரமேகங்
காதமெனு வோடக் கடத்துங்காண்- போத
மயக்க மொடுதாக மாறாச் சுரத்தின்
தயக்கமறுக் கும் மருதஞ் சாற்று.

குட்டரோக கங்கிருமி கோர வயிற்றுவலி
துட்டவறட் சூலை தொலையுங்காண்- சிட்டிப்
பொருதம்பா மென்னு விழிப் பூவையரே! நாளு
மருதம்பா ரென்றளவில் மாய்ந்து.

-அ.கு.

Botanical Name	:	Terminalia arjuna (Roxb)
Family	:	Combretaceae
Suvai	:	Thuvarppu
Thanmai	:	Thatpam
Pirivu	:	Karppu
Part Used	:	Bark
Properties	:	Tonic, Cardiac stimulant.

7.புளியம் பட்டை

புளிய மரத்தின் புரணிதனைக் கண்டால்
உளைமாந்தை குன்மவலி யோடும்- வளியார்ந்த
பேதிகட் டும்சுரம்போம் பித்தமுறுந் தீபனமாம்
ஒதஅ சீரணமே துன்னு.

-ப.கு.சி(149)

Botanical Name	:	Tamarindus indica, Linn
Family	:	Caesalpiniaceae
Suvai	:	Thuvarppu
Thanmai	:	Veppam
Pirivu	:	Karppu
Part Used	:	Bark
Properties	:	Mild astringent, tonic.

8.சீந்தில் கொடி

அமுதவல் லிக்கொடி யக்கார முண்டிடக்
திமிருறு மேகநோய்த் தீயெலா மாறுமே.

மேகமெனு மாதபத்தால் வெந்த வுயிர்ப்பயிரைத்
தாக மடங்கத் தணித்தலால்- ஆகம்
அமர ரெனலிருக்க வாதரித்த லாலே
அமுதவல்லி சஞ்சீவி யாம்.

-தேரன் வெண்பா.

Botanical Name	**:**	Tinospora cordifolia (Willd)
Family	**:**	Menispermaceae
Suvai	**:**	Kasappu
Thanmai	**:**	Veppam
Pirivu	**:**	Kaarppu
Part Used	**:**	Stem
Properties	**:**	Alterative, anti periodic, aphrodisiac, demulcent, stimulant, Stomachic, tonic, mild diuretic

9.கற்றாழை வேர்

பொல்லாமே கங்கபம்பு முச்சூலை குட்டம்ரசம்
அல்லார்மத் தம்பகந்த ரங்குன்மம் எல்லாம்விட்
டேகு மரிக்கு மெரிச்சற் கிரிச்சரமு
மாகு மரிக்கு மருண்டு.

-அ.கு.

Botanical Name	**:**	Aloe barbadensis, Miller
Family	**:**	Liliaceae
Suvai	**:**	Siru kasappu
Thanmai	**:**	Thatpam
Pirivu	**:**	Enippu
Part Used	**:**	Root
Properties	**:**	Tonic, alterative, purgative, emmenagogue

10.நீர்ப்பூலா வேர்

மாந்தங்க ணம்பொருமல் மாறாச் சலத்துடனே
சேர்ந்த சொறிசிரங்குந் தீருங்காண்- ஏந்தழிலைச்
சேர்ப்பாக கொண்ட செழுந்திருவே! பூவிலுறை
நீர்ப்பூலாப் பூண்டை நினை.

-அ.கு.

Botanical Name	**:**	Phyllanthus reticulates, Poir
Family	**:**	Euphorbiaceae
Suvai	**:**	Enippu, thuvarppu
Thanmai	**:**	Thatpam
Pirivu	**:**	Enippu
Part Used	**:**	Root
Properties	**:**	Alterative, refrigerant, diuretic.

11.நாரத்தை வேர்

நன்றி யுறவுலகில் நாரத்தங்காய் அருந்த
வென்றி தரும்புளிப்பால் மெய்ச்சுத்தம்- அன்றியுமோ
வாதமொடு குன்மமறும் வாற்கிருமி யும்போகும்
காதலுறு தீபனமாங் காண்.

-ப.கு.சி(202)

Botanical Name	**:**	Citrus medica, Linn
Family	**:**	Rutaceae
Suvai	**:**	Thuvarppu
Thanmai	**:**	Thatpam
Pirivu	**:**	Kaarppu
Part Used	**:**	Root
Properties	**:**	Astringent

12.நன்னாரி வேர்

சலதோடம் பித்தமதி தாகம் உழலை
சலமேறு சீதமின்னார் தஞ்சூ- டுலகமதிற்
சொன்னமது மேகம்புண் சுரமிவையெ லாமொழிக்கும்
மென்மதுர நன்னாரி வேர்.

-தே.கு

Botanical Name	**:**	Hemidesmus indicus, Linn R.Br
Family	**:**	Periplocaceae
Suvai	**:**	Enippu, siru kasappu
Thanmai	**:**	Thatpam
Pirivu	**:**	Enippu
Part Used	**:**	Root
Properties	**:**	Alterative, tonic, demulcent, diuretic, diaphoretic.

13.நத்தைச்சூரி (குழிமீட்டான்) வேர்

கணத்திலெழு மாந்தத்தைக் காணாவுட் சூட்டைக்
கணத்திலே வேரைக் களையும்- மனித்தமுலைப்
பாலைச் சுரப்பிக்கும் பாரிற் குழிமீட்டான்
காலைத் தொழுதுண்ணுங் கால்.

-ப.கு.சி.

Botanical Name	:	Spermacose hispida, Linn
Family	:	Rubiaceae
Suvai	:	Enippu, thuvarppu
Thanmai	:	Thatpam
Pirivu	:	Enippu
Part Used	:	Root
Properties	:	Alterative, tonic

14.சிறுகீரை வேர்

கண்புகைச்ச நேத்திரநோய் காசம் படலம்
புண்கிரிச்ச ரஞ்சோபை பொங்குபித்த- மண்பரவு
தாவரவிடங்களும் போம் தாழாத் திருவுமுண்டாம்
கூறுசிறு கீரைதனைக் கொள்.

-அ.கு.

Botanical Name	:	Amaranthus tricolor, Linn
Family	:	Amaranthaceae
Suvai	:	Enippu
Thanmai	:	Thatpam
Pirivu	:	Enippu
Part Used	:	Root
Properties	:	Diuretic, refrigerant, laxative

15.தாளிசபத்திரி

நாசி களப்பிணிகள் நாட்பட்ட காசஞ்சு
வாசம் அருசி வமனம்கால்- வீசிவரு
மேகமந்தம் அத்திசிரம் விட்டேகுந் தாளிச்சத்தால்
ஆகுஞ் சுகப்பிரச வம்.

-அ.கு.

Botanical Name	:	Taxus baccata, Linn
Family	:	Taxaceae
Suvai	:	Kaarppu
Thanmai	:	Veppam
Pirivu	:	Kaarppu
Part Used	:	Leaf
Properties	:	Stomachic, carminative, expectorant, tonic.

16.சுக்கு

சூலைமந்தம் நெஞ்செரிப்பு தோடமேப் பம்மழலை
மூலம் இரைப்பிருமல் மூக்குநீர்- வாலகப
தோடமதி சாரந் தொடர்வாத குன்மநீர்த்
தோடம்ஆ மம்போக்குஞ் சுக்கு.

-அ.கு

Botanical Name	**:**	Zingiber officinale, Rosc
Family	**:**	Zingiberaceae
Suvai	**:**	Kaarppu
Thanmai	**:**	Veppam
Pirivu	**:**	Kaarppu
Part Used	**:**	Rhizome
Properties	**:**	Stimulant, stomachic, carminative.

17.மிளகு

சீதசுரம் பாண்டு சிலேத்மங் கிராணிகுன்மம்
வாதம் அருசிபித்தம் மாமூலம்- ஓதுசன்னி
யாசமபஸ் மாரம் அடன்மேகம் காசமிவை
நாசங் கறிமிளகினால்.

-அ.கு.

கோணுகின்ற பக்கவலி குய்யவுரோ கம்வாத
சோணிதங்க முத்திற்குள் தோன்றுநோய்- காணரிய
காதுநோய் மாதர்குன்மங் காமாலை மந்தமென்றீர்
ஏதுநோய் காயிருக்கில் ஈங்கு.

-தே.கு.

தீயாகி யெங்கும் திரியுமதை யாவத்து
மோயாம லெப்படியு முண்டாக்காற்- பாயாது
போந்திமிர்வா தங்கிரந்தி புண்ணீரும் மண்ணவர்கும்
காந்திமெய்வா தச்சலுப்பைக் காய்.

-தேரண் வெண்பா.

Botanical Name	**:**	Piper nigrum, Linn
Family	**:**	Piperaceae
Suvai	**:**	Kasappu, kaarppu
Thanmai	**:**	Veppam
Pirivu	**:**	Kaarppu
Part Used	**:**	Dried fruit
Properties	**:**	Acrid, carminative, anti periodic, rubefacient, stimulant, resolvent, antidote, antivatha.

18.திப்பிலி

இருமல் குன்மம் இரைப்பு கயப்பிணி
ஈளை பாண்டு சந்யாசம் அரோசகம்
பொருமல் ஊதை சிரப்பிணி மூர்ச்சைநோய்
பூரிக் குஞ்சல தோடம் பிலீகமும்
வரும லப்பெருக் கோடு மகோதரம்
வாதம் ஆதிமுத் தோடஞ் சுரங்குளிர்
பெருமாலைப்புரி மேகம் பிடகமும்
பேருந் திப்பிலிப் பேரங்குரைக்கவே.

ஆசனநோய் தொண்டைநோய் ஆவரண பித்தமுதல்
நாசிவிழி காதிவைநோய் நாட்புழுநோய்- வீசிடுவி
யங்கலாஞ்ச னஞ்சிதையும் அம்பாய் அழிவிந்தும்
பொங்கலாஞ்ச நங்கையர்கோட் போல்.

-தே.கு.

கட்டி யெதிர்நின்று கடுநோயெல் லாம்பணியும்
திட்டி வினையகலும் தேகமெத்த- புட்டியாம்
மாமனுக்கு மாமனென மற்றவர்க்கு மற்றவனாங்
காமமெனுந் திப்பிலிக்கும் கை.

ஈளை யிரும லிரைப்புப் பசப்பிணிகள்
மாள வொழியாமல் வாட்டுமே- யாளுமுறை
பாங்கா யற்ந்துசெய்வீர் பண்டிதத்தைப் பண்டிதரே
வேங்கைவாய்ப் பான்கணை மெய்.

-தேரண் வெண்பா.

Botanical Name	**:**	Piper longum, Linn
Family	**:**	Piperaceae
Suvai	**:**	Kaarppu
Thanmai	**:**	Veppam
Pirivu	**:**	Enippu
Part Used	**:**	Dried fruit
Properties	**:**	Stimulant, carminative.

19.இலவங்கம்

பித்த மயக்கம் பேதியொடு வாந்தியும்போம்
சுத்தவிரத் தகடுப்புந் தோன்றுமோ- மெத்த
இலவங்கங் கொண்டவருக் கேற் சுகமாகும்
மலமங்கே கட்டுமென வாழ்த்து.

சுக்கிலநட் டங்கர்ண் சூர்வியங்க லாஞ்சனந்தாட்
சிக்கல்விடாச் சர்வா சியப்பிணியுந்- மக்கிக்குட்
டங்கப் பூவோடுவ் தரிபடருந் தோன்றிலில்
வங்கப்பூ வோடுரைத்து வா.

-அ.கு.

Botanical Name	:	Syzygium aromaticum, (Linn) Merrill & Perry
Family	:	Myrtaceae
Suvai	:	Kaarppu viruviruppu
Thanmai	:	Veppam
Pirivu	:	Kaarppu
Part Used	:	Dried bud
Properties	:	Antispasmodic, carminative, stomachic

20.கோரைக் கிழங்கு

சீத சுரந்தீர்குஞ் செம்புனல்பித் தம்போகும்
வாத சுரந்தணிக்கும் வையகத்தில்- வேதைசெய்
வந்த பிணியையெல்லாம் வாட்டுமுத் தக்காசு
கொந்துலவும் வார்குழலே! கூறு.

அதிசாரம் பித்தம் அனற்றாகம் ஐயங்
குதிவாதஞ் சோபங் கொடிய- முதிர்வாந்தி
யாரைத் தொடர்ந்தாலும் அவ்வவர்க்கெ லாங்குளத்துக்
கோரைக் கிழங்கைக் கொடு.

-அ.கு.

Botanical Name	:	Cyperus rotundus, Linn
Family	:	Cyperaceae
Part Used	:	Root tuber
Properties	:	Astringent, stimulant, tonic, diuretic, diaphoretic, demulcent, emmenagogue, vermifuge.

21.பெருங்காயம்

தந்தவே தந்த மூலத்தெழும்பிணி
சருவகாளம் விருச்சிகங்கீடம்மா
மந்தம்வாதம் உதாவர்த்தம் அல்குல்நோய்
மார்பணங்கட்ட குன்மம்மகோதரம்
உந்துகெர்ப்பத்தின் வித்திரஞ்சூலைச்சூர்
உதிரப்பூச்சி சிலேத்துமத்துறும்வலி
வந்தமெய்க்கடுப் போடிவைமுற்றுமே
மாயுநாறுநற் காயங்கிடைகினே.

-தே.கு.

Botanical Name	:	Ferula asafoetida, Linn
Family	:	Apiaceae
Suvai	:	Kasappu, karakarappu
Thanmai	:	Veppam
Pirivu	:	Kaarppu

Part Used	:	Gum-oleoresin
Properties	:	Stimulant, carminative, antispasmodic, expectorant, laxative, anthelmintic, diuretic, aphrodisiac, emmenagogue

22.சாதிப்பத்திரி

சாதிதரும் பத்திரிக்குத் தாபச் சுரந்தணியும்
ஓதுகின்ற பித்தம் உயருங்காண்- தாதுவிர்த்தி
யுண்டாங் கிராணியோ டோதக் கழிச்சலறும்
பண்டாங் குறையே பகர்.

-அ.கு.

Botanical Name	:	Myristica fragrans, Houtt
Family	:	Myristicaceae
Suvai	:	Kaarppu, thuvarppu
Thanmai	:	Veppam
Pirivu	:	Kaarppu
Part Used	:	Aril
Properties	:	Aphrodisiac, carminative, stimulant, hypnotic.

RAW DRUG PHOTOS

		
Atthi - Ficus recemosa	**Kondrai - Cassia fistula**	**Aavarai - Cassia auriculata**
		
Kadalalinjil - Salacia reticulate	**Iluppai - Madhuca longifolia**	**Puli - Tamarindus indica**
		
Maruthu - Terminalia arjuna	**Nathaisoori - Spermacoce hispida**	**Naarathai - Citrus medica**
		
Nannaari - Hemidesmus indicus	**Sirukeerai - Amaranthus tricolor**	**Neerpoola - Phyllanthus reticulates**

		
Kattralai - Aloe barbadensis	**Korai - Cyperus rotundus**	**Seenthil - Tinospora cordifolia**
		
sukku - Zingiber officinale	**Milaku - Piper nigrum**	**Thippili - Piper longum**
		
Thalisa pathri - Abies spectabilis	**Saathi pathri - Myristica fragrans**	**Kirambu - Syzygium aromaticum**
		
Perungaayam - Ferula asafoetida		

STANDARD OPERATING PROCEDURE

SOURCE OF RAW DRUGS:

The required raw drugs for preparation of ***ATTHIPPATTAIYATHI KASAYAM*** are purchased from a well reputed country shop. The raw drugs will be authenticated by the Botanist, Medicinal Botany Department of NIS. The raw drugs are purified and medicine is prepared in *Gunapadam* lab of National Institute of Siddha. The prepared medicine is again authenticated by the HOD of Gunapadam department, NIS.

PURIFICATION METHODS:

1. **Roots: (General method)**
    a. Wash the roots with running water and dry it.
2. **Barks: (General method)**
    a. Clean the barks with cotton cloth and remove the peel with small knife and dry it.

3. **Kadalazhinjil -** ***Salacia reticulate, Wight***

a. Dry it in sunlight.

4. **Korai kizhangu –** ***Cyperus rotundus,Linn.***

a. Dry it in sunlight.

5. **Seenthil -** ***Tinospora cordifolia (Willd)***

a. Remove the peel and dry it.

6. **Sukku –** ***Zingiber officinalis,Rosc***

a. Double the proportion of lime stone [calcium carbonate] solution is poured and boiled for three hours, then wash it, dry and remove the peel.

7. **Milagu –** ***Piper nigrum,Linn.***

a. Soak it in sour butter milk for three hours.

8. **Thippili –** ***Piper longum,Linn.***

a. Soak it in plumbago zeylanica,Linn leaf(Ceylon lead wort- Venkodiveli) juice for twenty four minutes (1 Nazhigai) and dry it in sunlight.

9. **Thalisa pathri -** ***Abies spectabilis, (D.Don) Mirb***

a. Dry it in sunlight.

10. **Saathi pathri -** ***Myristica fragrans, Houtt***

a. Dry it in sunlight.

11. **Kirambu-** ***Syzygium aromaticum, (Linn) Merrill & Perry***

a. Dry it in sunlight.

12. **Perungaayam -** ***Ferula asafoetida,Linn***

a. Cut into small pieces and roasted it or Soak it in Nelumbo nucifera, Gaertn leaf(Lotus- Thamarai) juice for twenty four minutes (1 Nazhigai) and dry it.

METHOD OF PREPARATION

Kasayam powder preparation method:

All the purified drugs are ground into a coarse powder by using iron mortar & pestle.

Kasayam preparation method:

Take 5gm (*verukadi*) of *Atthippattaiyathi kasayam* powder and add 1.3 lit (*nazhi*) of water, boil it to reduce 1/4th of the part, ie 336ml (*uzhakku*).

DRUG STORAGE:

The trial drug, ***Atthippattaiyathi kasayam*** powder is stored in clean and dry wide mouthed glass bottles.

DISPENSING:

The study drug packages will contain 150gm of *ATTHIPPATTAIYATHI KASAYAM* powder sachets. At each visit (once in 10 days for 40 days) the patients will be given the above drug packages for 10 days of treatment. At each visit the patients will be advised to bring back the unconsumed drugs and return to the research scholar.

DOSAGE:

- Kasayam powder -5gm *(verukadi*)
- Kasayam- 336ml *(uzhakku*) 3 times per day.

COURSE: 40 days

INDICATIONS: Neerizhivu (Mathumeagm)

REFERENCE: Agathiyar 2000- 3rd volume, Author: Dr.S.Venkattarajan, L.I.M, 5th Edition- Oct 2002. Page No: 4, 5. Publisher: Saraswathy Mahal Library, Thanjavur.

ATTHIPPATTAIYATHI KASAYAM PHOTOS

Atthippattaiyathi kasayam powder

Atthippattaiyathi kasayam (Decoction)

CLINICAL STUDY

STUDY DESIGN & CONDUCT OF STUDY

Study Type: An open clinical trial.

Study place: OPD and IPD of Ayothidoss Pandithar Hospital, National Institute of siddha, Tambaram sanatorium, Chennai-47.

Study period: 12 months

Sample size: 40 patients

SUBJECT SELECTION

As and when patients' reporting at OPD of Ayothidass Pandithar Hospital with symptoms of inclusion criteria was subjected to screening test & documented using screening proforma.

INCLUSION CRITERIA

- Age : 30-55Yrs
- Sex: Male & Female

Symptoms:

- Polyuria
- Nocturia
- Polydipsia
- Polyphagia
- Body pain
- Weight gain (obesity), Tiredness, Burning feet and genital pruritus.

Blood glucose level:

- Fasting plasma glucose level- 126 to180 mg/dl
- 2 hours postprandial plasma glucose level- 200 to 300 mg/dl

Asymptomatic individuals fulfilling the following criteria may be screened

- Previously identified Impaired Fasting Glucose (IFG) or Impaired Glucose Tolerance (IGT).
  - IFG- FPG >110 and <126mg/dl
  - IGT- 2h PG >140 and <200mg/dl
- Over weight – Body mass index $\geq 23kg/m^2$
- Family history of diabetes
- Sedentary lifestyle
- History of gestational diabetes mellitus, recurrent fetal loss or delivery of large baby $\geq 3.5kg$
- Dyslipidemia

- Hypertension (>140/90 mm hg in adults)
- Urine test – glycosuria, microalbuminuria
- Acanthosis nigricans
- Patient willing to sign the informed consent stating that he will conscientiously stick to the treatment during 40 days but can opt out of the trial of his own conscious discretion.
- Patients who are willing to provide blood and urine for lab investigation.

EXCLUSION CRITERIA

- IDDM (Insulin Dependent Diabetes Mellitus)
- Diabetic complications like microvascular and macrovascular complications etc.
- Cardiac diseases
- Pulmonary diseases
- Renal diseases
- Thyroid dysfunctions
- Gestational diabetes
- Other endocrine abnormalities
- Patient who are not willing to give blood sample

WITHDRAWAL CRITERIA

- Intolerance to the drug & development of any serious adverse reactions during drug trial.
- Poor patient compliance & defaulters.
- Patient turned unwilling to continue in the course of clinical trial.
- Increase in severity of symptoms.
- Uncontrolled blood sugar level

TEST & ASSESSMENTS

1. **CLINICAL ASSESSMENT**
2. **SIDDHA ASSESSMENT**
3. **ROUTINE INVESTIGATION**
4. **SPECIFIC INVESTIGATION**

1. CLINICAL ASSESSMENT

- Increased frequency of Urination (polyuria)
- Thirst (polydipsia)
- Excessive hunger (polyphagia)

- Body pain
- Tiredness
- Burning feet
- Generalized/genital pruritus
- Dull pain in the testis
- Yellow coloured urine

2. SIDDHA ASSESSMENT

Thinai (Living Place)

Paruvakaalam (Season)

Gnanenthiriyam and Kanmenthiriyam:

1. Vaai (Buccal Cavity)
2. Kaal (lower limb)
3. Kai (upper limb)
4. Eruvaai (anorectal region)
5. Karuvaai (uro- genital region)

Ezhu Udal Kattugal:

1. Saram
2. Senneer
3. Uoon
4. Kozhuppu
5. Enbu
6. Moolai
7. Sukkilam/Suronitham

Enn Vagai Thervu (Eight types of Examination):

1. Nadi (Pulse perception)
2. Naa (Tongue)
3. Niram (Complexion)
4. Mozhi (Voice)
5. Vizhi (Eyes)
6. Parisam (Palpatory perception)
7. Malam (Bowel habits)
8. Moothiram (Urine){Neerkuri& Neikuri}

3. ROUTINE INVESTIGATION

HAEMATOLOGY

- Hb (gms %)
- Total WBC Count(cells/cumm)
- DC
    - Polymorphs (%)
    - Lymphocytes (%)
    - Eosinophils (%)
    - Monocytes (%)
    - Basophils (%)
- Total RBC count (cells/cu.mm)
- ESR (mm/hr)

CLINICAL BIOCHEMISTRY

RENAL FUNCTION TEST

- Blood urea (mg/dl)
- S. total creatinine (mg/dl)
- Uric acid (mg/dl)

LIPID PROFILE

- S. Total cholesterol (mg/dl)
- HDL (mg/dl)
- LDL (mg/dl)
- VLDL (mg/dl)
- TGL (mg/dl)

LIVER FUNCTION TEST

- S. Total bilirubin (mg/dl)
- S. Direct bilirubin (mg/dl)
- S. Indirect bilirubin (mg/dl)
- SGOT (U/dl)
- SGPT (U/dl)
- S. Alkaline phosphatase (U/dl)
- S. Total protein (g/dl)
- S. Albumin (g/dl)
- S. Globulin (g/dl)

OTHER TEST

- S. Calcium (mg/dl)
- S. Phosphorous (mg/dl)

URINE EXAMINATION

- Neerkuri & Neikuri
- Albumin
- Sugar (Fasting & postprandial)
- Deposits

SIDDHA PARAMETERS

- Neerkuri and Neikuri
- Malam

4. SPECIFIC INVESTIGATION

- OGTT

- Fasting (over night fast)
- 2 Hours after glucose load

STUDY ENROLLMENT

- In this clinical trial, patients reporting at NIS OPD with the clinical symptoms of Polyuria, Polydipsia, Polyphagia, General body pain and tiredness was examined clinically for enrolling in the study based on the inclusion and exclusion criteria.
- The patients who are to be enrolled in this study was informed (Form IV) about the objective of the study, trial drug, possible outcomes in their own language and terms understandable to them.
- After ascertaining the patient's willingness, informed consent was obtained in the consent form (Form IV A).
- All these patients was given unique registration card in which patients' Registration number of the study, Address, Phone number and Doctors phone number etc. so as to report adverse reaction.
- Complete clinical history, complaints and duration, examination findings all was recorded in the prescribed Proforma in the history and clinical assessment forms separately. Screening Form- I was filled up; Form I-A, Form –II and Form –III was used for recording the patient's history, clinical examination of symptoms and signs and laboratory investigations respectively.
- Patients were advised to take the trial drug and appropriate dietary advice (Form IV-E) would be given according to the patients' perfect understanding.

CONDUCT OF THE STUDY:

The trial drug ATTHIPPATTAIYATHI KASAYAM was given continuously for 40 days for OP patients, they should visit the hospital once in 10 days. At each clinical visit clinical assessment was done and prognosis was recorded. For IP patients the drug is provided daily and prognosis was noted. For IP patients' also clinical assessment was done daily. Laboratory investigations were done 0^{th} day & 40^{th} day of the trial. For IP patients, who was not in a situation to stay in the hospital for 40 days were advised to attend the OPD for further continuation of course of treatment. After the end of the treatment also, the patient was advised to visit the OPD for another 2 months for follow-up. If any trial patient who fails to collect the trial drug on the prescribed day but wants to continue in the trial from the next day or two, he/she was allowed, but defaulters of one week and more was not allowed to continue and withdrawn from the study with fresh case being inducted.

DATA MANAGEMENT

- After enrolling the patient in the study, a separate file for each patient was opened and all forms was filed in the file. Study Number and Patient Number were entered on the top of file for easy identification. Whenever the study patient visits OPD during the study period, the respective patient file was taken and necessary recordings were made at the assessment form or other suitable form.
- The screening forms were filed separately.
- The Data recordings were monitored for completion by HOD and adverse event by Pharmacovigilance Department of National Institute of Siddha. All forms were further scrutinized in presence of Investigators by Senior Research Officer (Statistics) for logical errors and incompleteness of data to avoid any bias. No modification in the results is permitted for unbiased reports.

OUTCOME:

Primary outcome:

The outcome is mainly assessed by comparing the pre and post treatment blood glucose level of the trial patient.

Secondary outcome:

Secondary outcome is assessed by comparing the following parameters, pre and post treatment.

- Changes in siddha and clinical parameters
- Changes in investigation parameters

ADVERSE EFFECT/SERIOUS EFFECT MANAGEMENT:

If the trial patient develops any adverse reaction, it was recorded in Adverse Reaction Form and he/she was referred to the member of the Pharmacovigilance Department of NIS, and proper management was given by the investigator in OPD of NIS.

STATISTICAL ANALYSIS

All collected data were entered into computer using MS access / MS excel software by the investigator. The data was analyzed using STATA software under the guidance of SRO (stat), NIS. The level of significance was 0.05 Descriptive analyses were made and necessary tables/graphs generated to understand the profile of the patients included in the study. Student‘t’ test and chi-square test were proposed to be performed for quantitative and qualitative data.

ETHICAL ISSUES

1. Informed consent was obtained from the patient explaining in the understandable language to the patient.
2. After the consent of the patient (through consent form) they were enrolled in the study.
3. Treatment was provided free of cost.
4. No other external or internal medicines were used. There was no infringement on the rights of patient.
5. To prevent any infection, while collecting blood sample from the patient, only disposable syringes, disposable gloves, with proper sterilization of lab equipments was used.
6. The data collected from the patient was kept confidentially. The patient was informed about the diagnosis, treatment and follow-up.
7. In conditions of treatment failure, adverse reactions, Patients was given alternate treatment at National Institute of Siddha with full care throughout the end.
8. The patient who are excluded (as per exclusion criteria) are given proper treatment with full care at National Institute of Siddha.

ASSESSMENT FORMS

FORM I SCREENING & SELECTION PROFORMA

FORM I A HISTORY PROFORMA ON ENROLLMENT

FORM II CLINICAL ASSESSMENT ON ENROLLMENT

FORM II A CLINICAL ASSESSMENT DURING & AFTER TRIAL

FORM III LABORATORY INVESTIGATION ON ENROLLMENT & CONCLUSION OF TRIAL

FORM IV INFORMATION SHEET

FORM IV A CONSENT FORM

FORM IV B WITHDRAWAL FORM

FORM IV C DRUG COMPLIANCE FORM

FORM IV D DIETARY ADVICE FORM

FORM IV E ADVERSE REACTION FORM

Observations and Results are tabulated under the following headings

1. Age incidence.
2. Sex distribution
3. Socio-economic status
4. Family history
5. Food habits
6. Thinai
7. Paruva Kaalam
8. Yaakai
9. Mukkutrangal

9.a) Derangement in Vatham

9.b) Derangement in Pitham

9.c) Derangement in Kabam

10. Ezhu udal thaathukkal
11. a) Enn vagai thervugal

b) Niram

c) Manam

d) Nurai

e) Edai

f) Enjal

g) Neikuri

h) Naadi

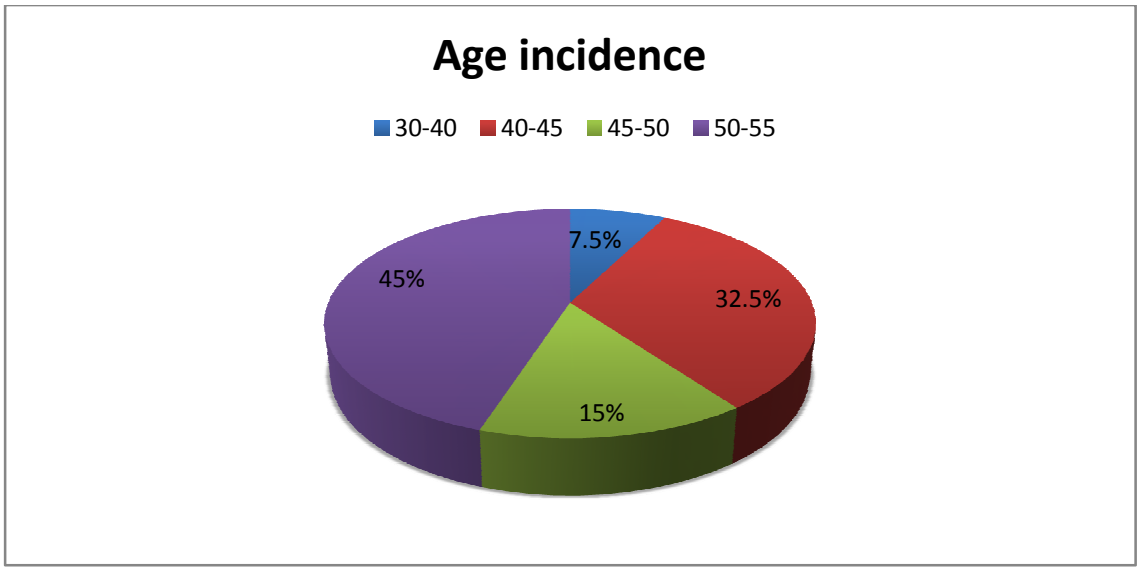
12. Clinical features
13. Chronicity of illness
14. Gradation of result
15. Prognosis of Clinical features

OBSERVATION AND RESULTS

Table-1

Age incidence

S.no	Age in year	No of cases	Percentage (%)
1.	30-40	3	7.5%
2.	41-45	13	32.5%
3.	46-50	6	15%
4.	**51-55**	**18**	**45%**
	Total	40	100%



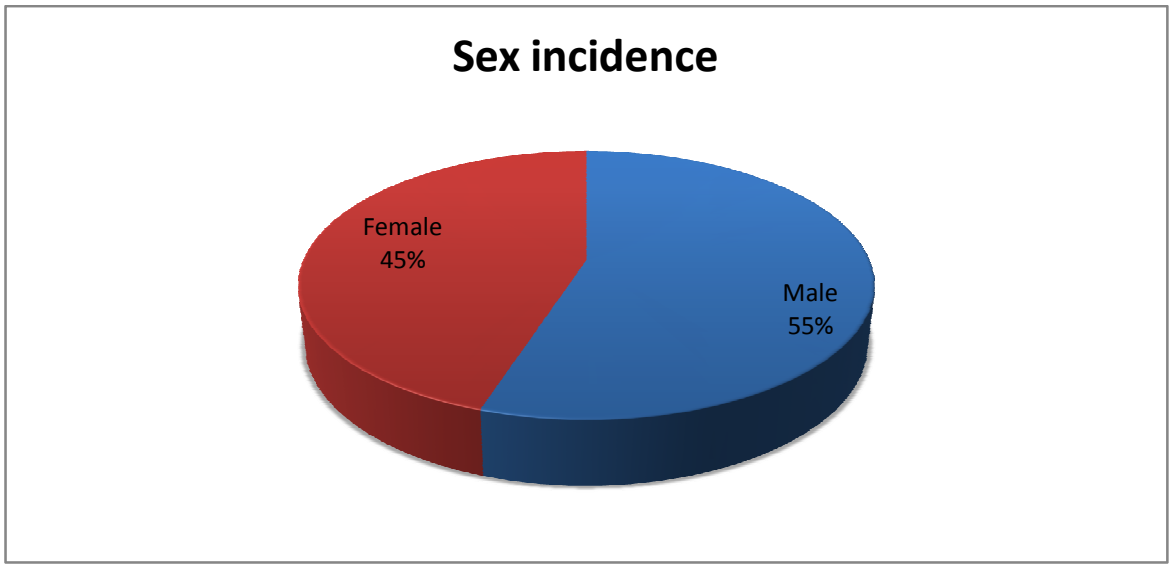
Observation:

As per table 1 & fig 1: The prevalence of the disease was found to be higher in 18 cases (45 %) in the age group of 51 - 55 years, 13 cases (32.5%) in the age group of 41 – 45 years, 6 cases (15 %) in the age group of 46 - 50 years and 3 cases (7.5 %) in the age group of 30 - 40 years.

Table-2

Sex incidence

S.no	Sex	No of cases	Percentage (%)
1	Male	22	55%
2	Female	18	45%
	Total	40	100%



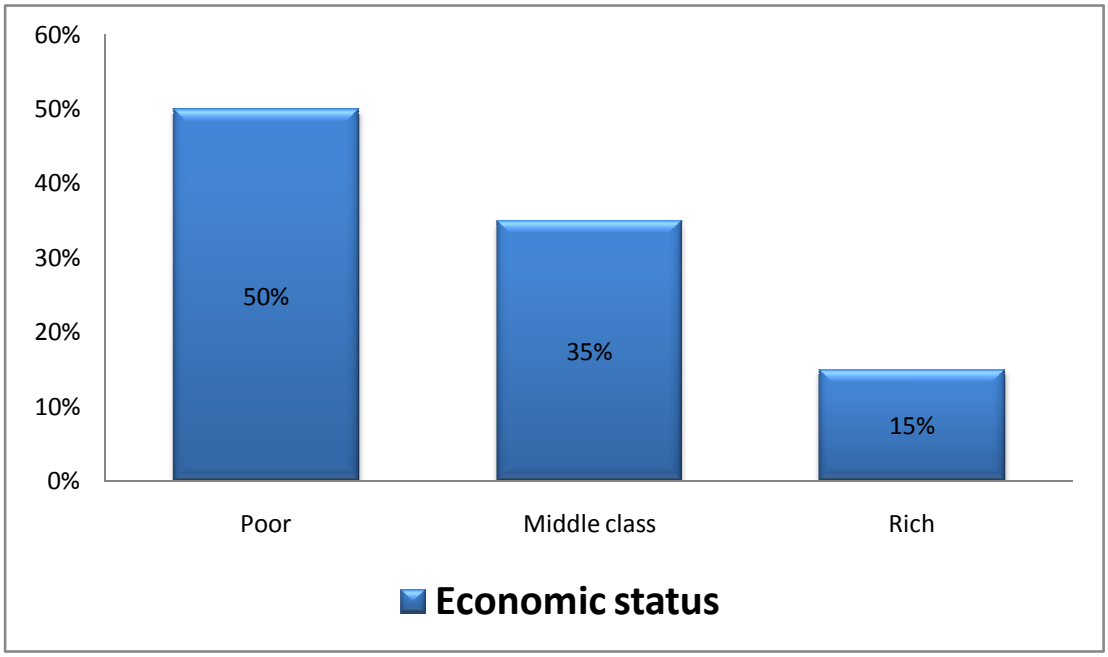
Observation:

As per table 2 and fig 2: Among the 40 patients selected, prevalence of the disease was found to be higher in males i.e. 22 cases (55%) then the Female cases of 18 (45%).

Table-3

Economic status

S.no	Economic status	No of cases	Percentage (%)
1.	Poor	20	50%
2.	Middle class	14	35%
3.	Rich	6	15%
	Total	40	100%



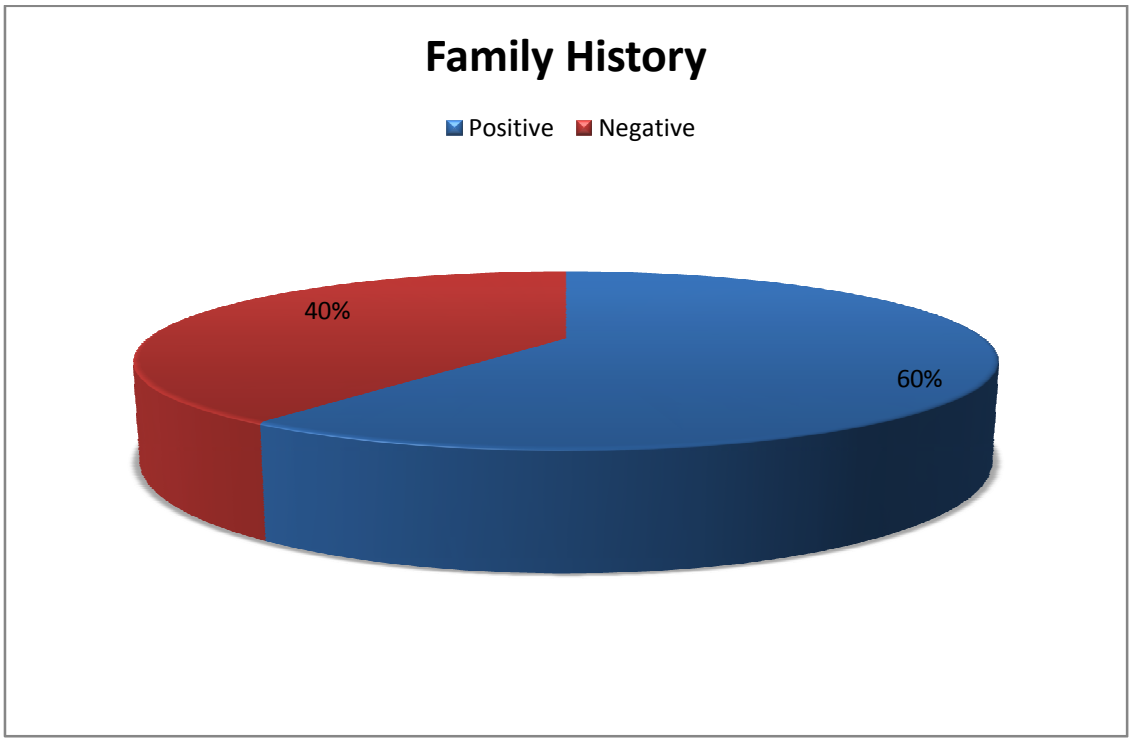
Observation:

As per table 3 and Fig 3: The incidence of the disease was found to be higher in 20 (50 %) cases belonging to poor class, medium in 14 (35 %) cases belonging to middle class and lower in 6 (15) cases belonging to high class.

Table-4

Family history

S.no	Family history	No. of cases	Percentage (%)
1.	Positive	24	60%
2.	Negative	16	40%
	Total	40	100%



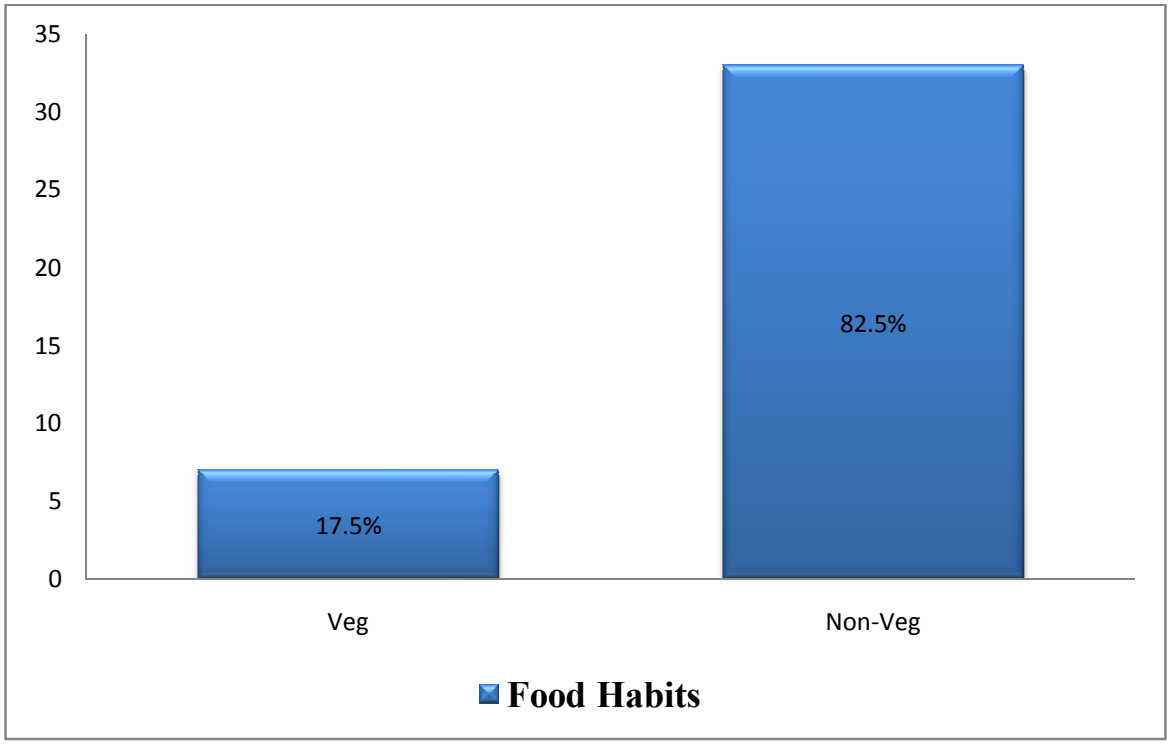
Observation:

As per table 4 and Fig 4. Among the 40 cases, Positive familial history was seen in 24 (60 %) patients and no history of family involvement was found in 16 cases(40 %).

Table-5

Food habits

S.no	Food habits	No. of cases	Percentage (%)
1.	Vegetarians	7	17.5%
2.	Non-vegetarians(Mixed)	33	82.5%
	Total	40	100%



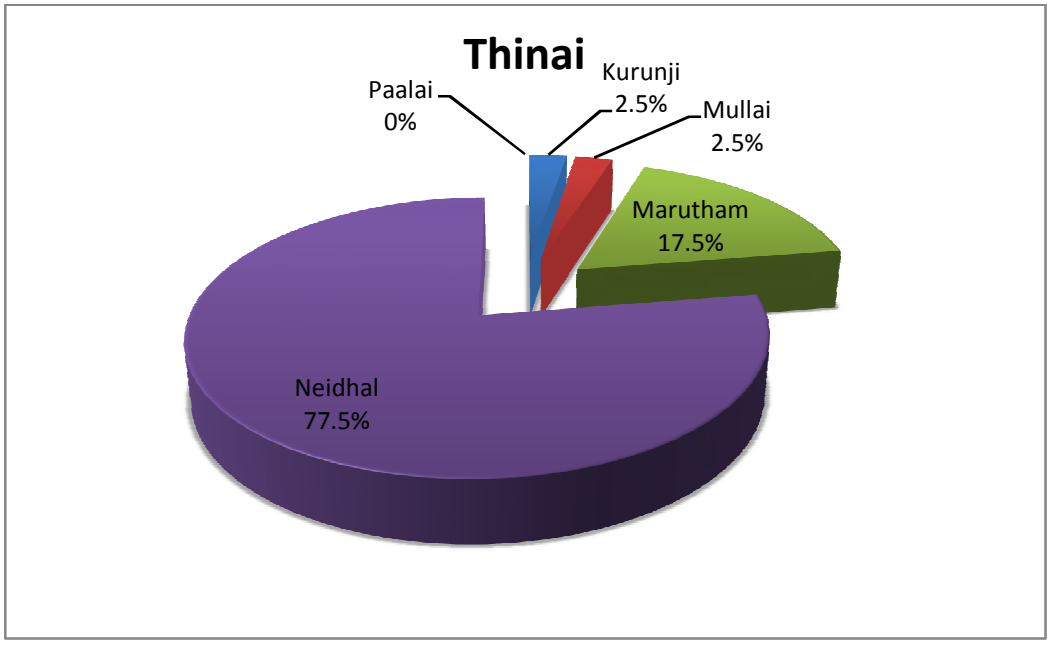
Observation:

As per table 5 and fig 5: Among 40 cases the incidence of the disease was higher in 33 (82.5%) cases, which were non vegetarians and lower in vegetarians 7 cases (17.5%).

Table-6

Distribution of *Thinai (Land)*

S. No	Thinai	No. of cases	Percentage (%)
1.	Kurinji	1	2.5%
2.	Mullai	1	2.5%
3.	Marutham	7	17.5%
4.	**Neidhal**	**31**	**77.5%**
5.	Paalai	0	0%
	Total	40	100%



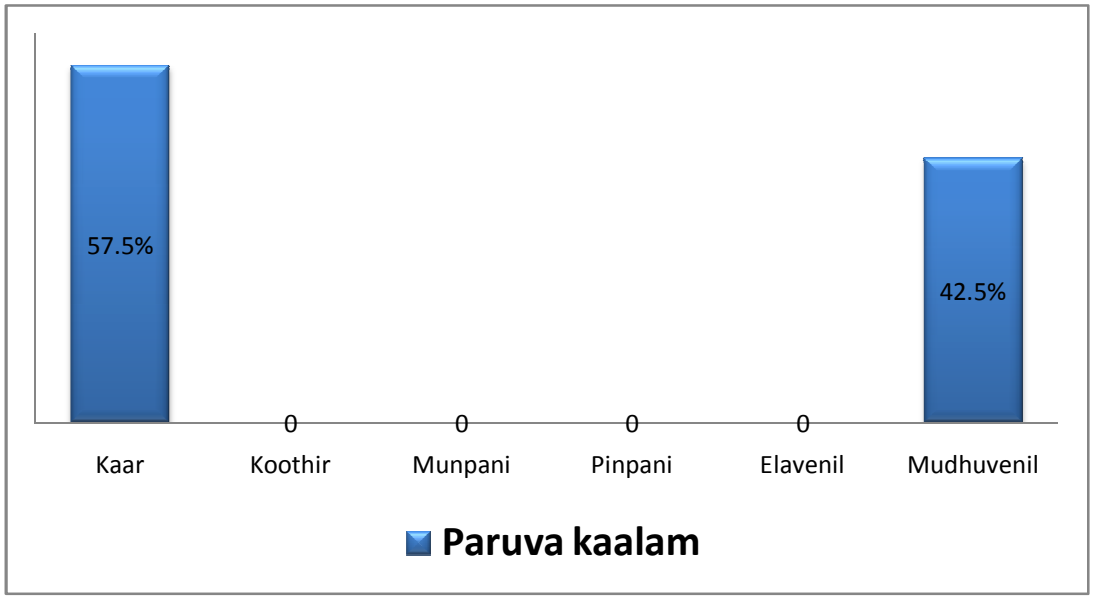
Observation:

As per table 6 and fig 6: Among 40 cases 31 (77.5%) cases belongs to Neidhal, 7 (17.5%) cases were from Marutham, and only one (2.5%) case was from Kurunji and Mullai.

Table-7

Paruva kaalangal (Season)

S.no	Paruva kaalam	Months	No. of cases	Percentage(%)
1.	**Kaar kaalam**	**Aavani- Puratasi** (Aug. 17 - Oct 17)	**23**	**57.5%**
2.	Koothir kaalam	Aipasi-karthigai (Oct. 18 - Dec. 15)	0	0
3.	Mun pani kaalam	Margazhi-Thai (Dec. 16 - Feb. 12)	0	0
4.	Pin pani kaalam	Masi- panguni (Feb.13 - Aprl. 13)	0	0
5.	Elavenil kaalam	Chithirai- Vaigasi (Aprl. 14 - June 14)	0	0
6.	Mudhuvenil kaalam	Aani- Aadi (June 15 - Aug. 16)	17	42.5%
		Total	40	100%



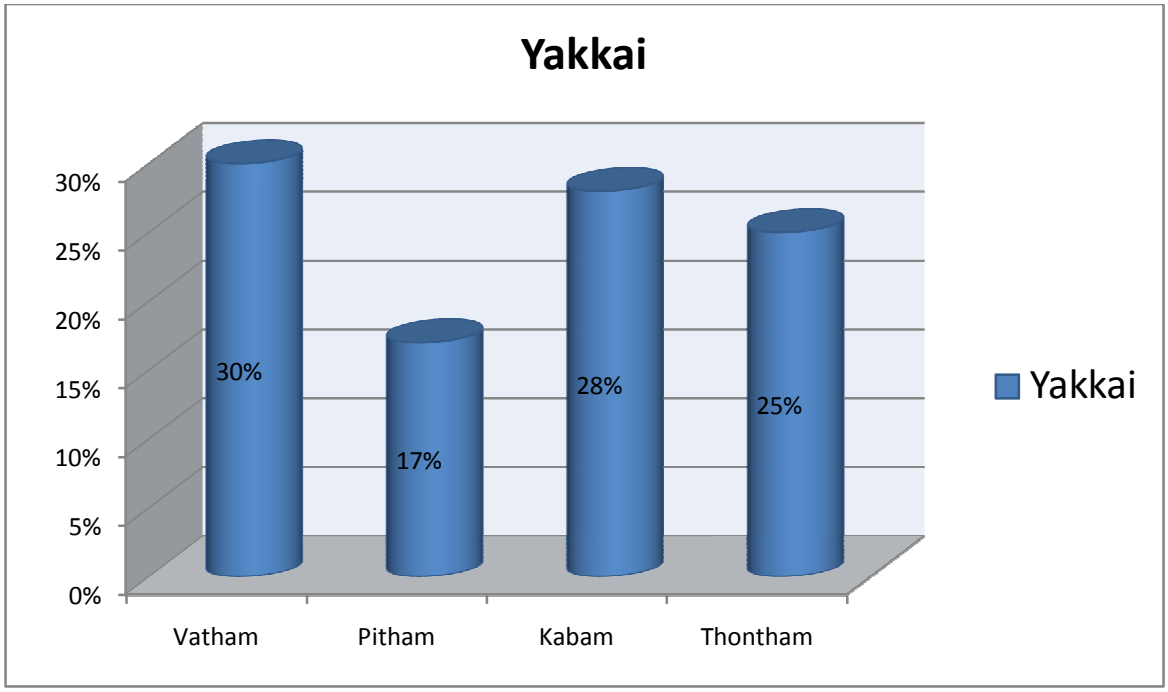
Observation:

As per table 7 and fig 7: Among the 40 cases, in 23 cases (57.5%) the incidence of the disease seems to be higher in Kaar kaalam (Avani-Puratasi), 17 cases (42.5%) in Muthuvenil kaalam (Aani-Aadi).

Table-8

Yaakkai

S. no	Yakkai	No. of cases	Percentage (%)
1.	Vaatham Thegi	12	30%
2.	Pittham Thegi	7	17%
3.	Kapham Thegi	11	28%
4.	Thontham Thegi	10	25%
	Total	40	100%



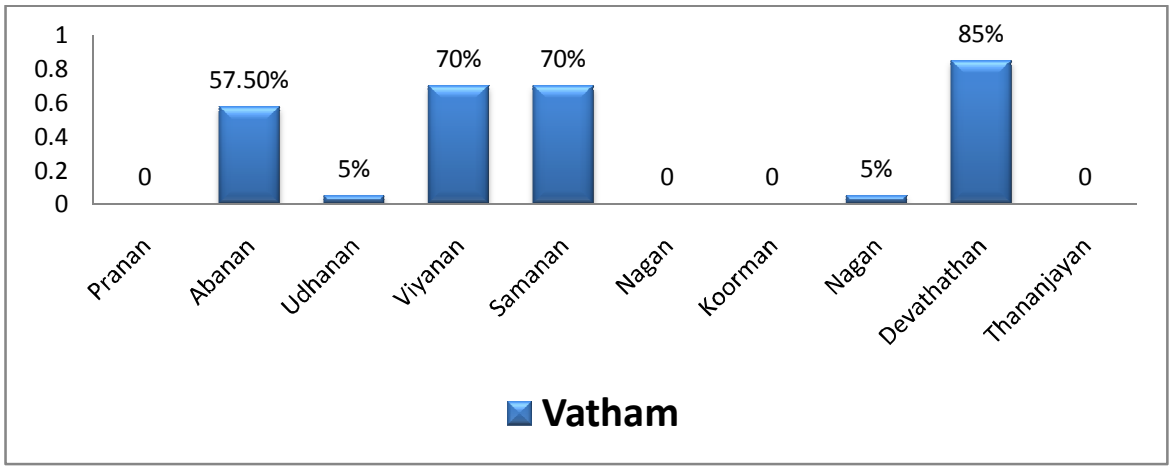
Observation:

As per table 8 and fig 8: Among the 40 cases, 12 cases (30%) were vaatha thegi, 7cases (17%) were pitha thegi, 11 cases (28%) were kappa thegi and 10 cases (25%) were thontha thegi.

Incidence according to Mukkutrangal

Table-9a (i) Derangement in Vaatham

Sl. No	Vaatham	No. of cases	Percentage (%)
1	Praanan	0	0
2	Abaanan	23	57.5%
3	Udhaanan	2	5%
4	Viyaanan	28	70%
5	Samaanan	28	70%
6	Naagan	0	0
7	Koorman	0	0
8	Kirukaran	2	5%
9	Devathaththan	34	85%
10	Thananjeyan	0	0

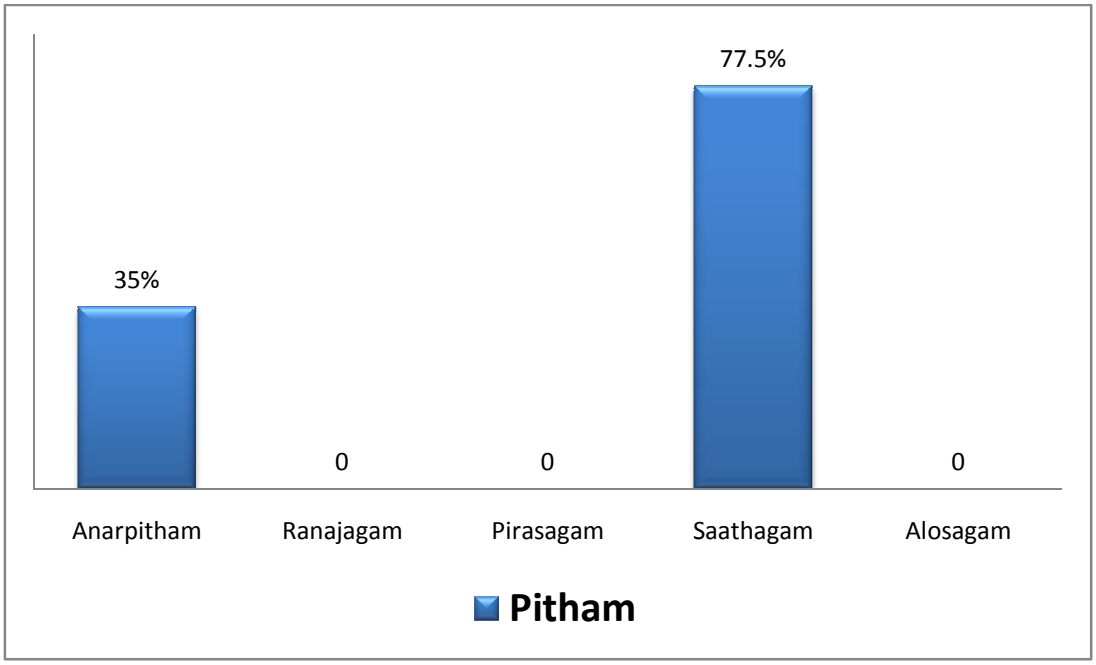


Observation:

As per table 9a and Fig 9a: Among 40 cases, Samanan and viyanan were affected in 28 (70%) cases. In 34 (85 %) cases Devathathan was affected. Abanan was affected in 23 (57.5%) cases and Udhanan was affected in 2 (5%) cases.

Table-9b (ii) Derangement in Piththam

S.no	Piththam	No. of cases out of 40	Percentage (%)
1.	Anarpitham	14	35%
2.	Ranjagam	0	0
3.	Pirasagam	0	0
4.	Saathagam	31	77.5%
5.	Aalosagam	0	0

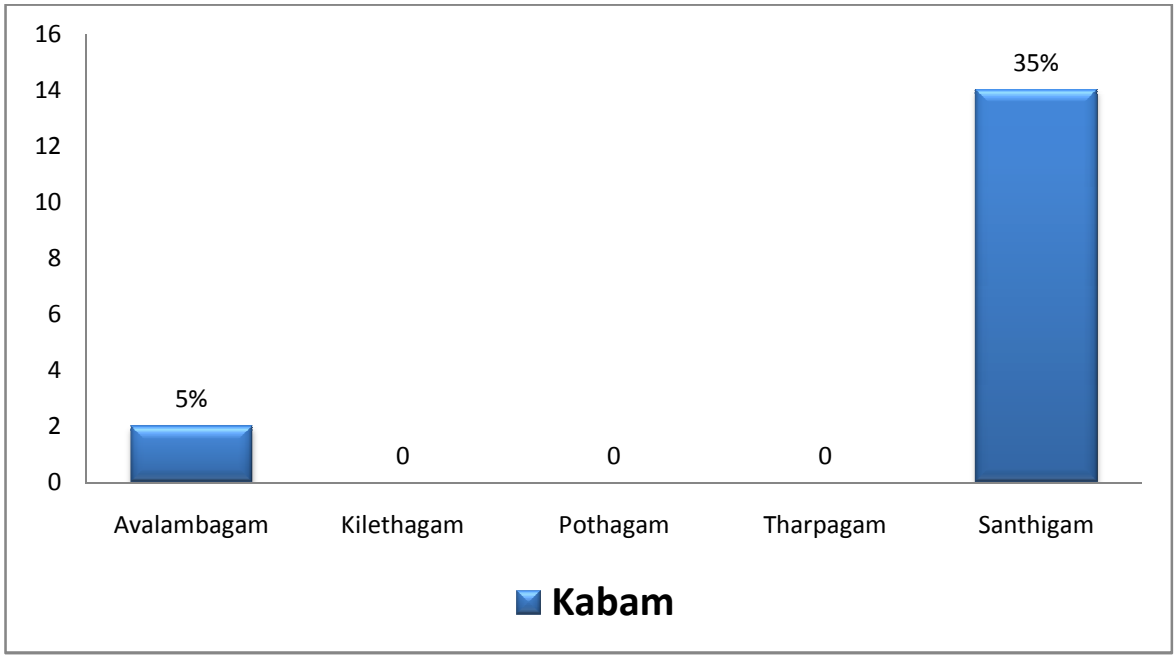


Observation:

As per table 9b and Fig 9b: Among the 40 cases, Sathagam was affected in 31 (77.5%) cases and Anarpitham was affected in 14 (35%) cases.

Table-9c (iii) Derangement in Kabam

S.no	Kapham	No. of cases out of 40	Percentage (%)
1.	Avlambagam	2	5%
2.	Kilethagam	0	0
3.	Pothagam	0	0
4.	Tharpagam	0	0
5.	Santhigam	14	35%



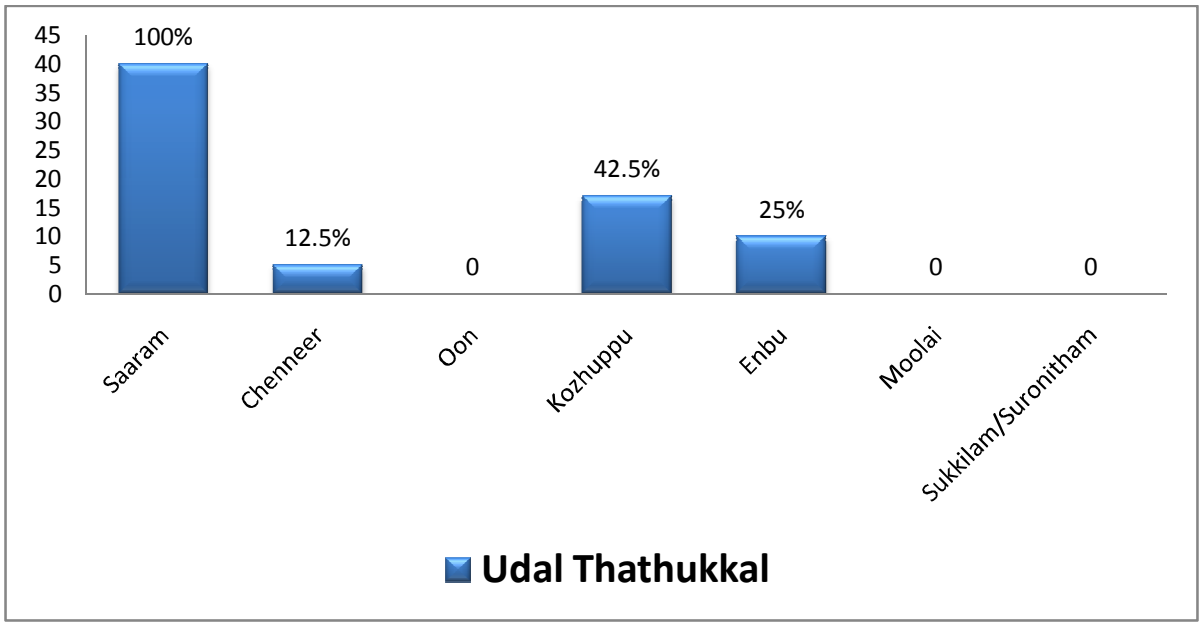
Observation:

As per table 9c and Fig 9c: Among 40 cases Avalambagam was affected in 2 (5%) cases and Santhigam was affected in 14 (35 %) as a result of pain in both lower limbs.

Table-10

Ezhu udal thaathukkal

S.no	Udal thaathukkal	No .of cases out of 40	Percentage (%)
1.	Saaram	40	100%
2.	Chenneer	5	12.5%
3.	Oon	0	0
4.	Kozhuppu	17	42.5%
5.	Enbu	10	25%
6.	Moolai	0	0
7.	Sukilam/Suronitham	0	0



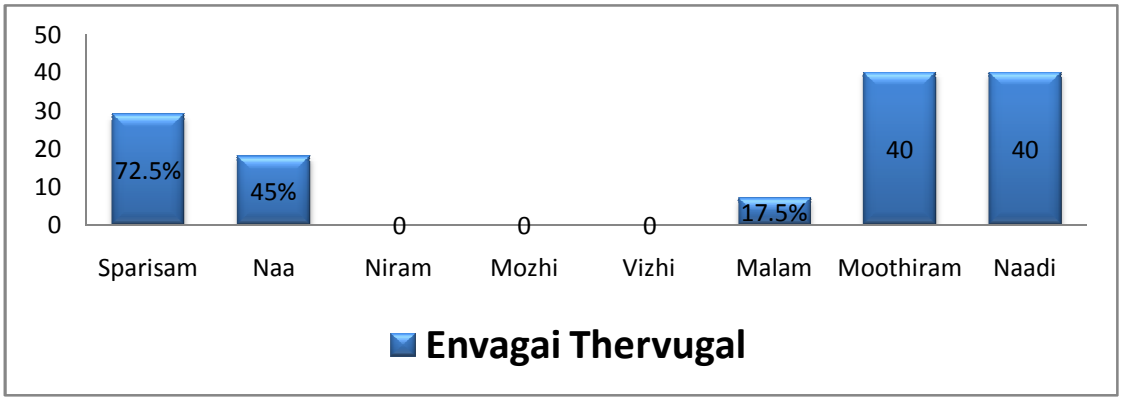
Observation:

As per table 10 and Fig 10: A mong 40 cases, Saaram was affected in all the 40 (100%) cases, Senner was affected in 5 (12.5 %) cases, Kozhuppu affected in 17 cases (42.5%) and Enbu affected in 10 cases (25%).

Table-11a

Envagai thervugal

S.no	Enn vagai thervugal	No. of cases out of 40	Percentage (%)
1.	Sparisam	29	72.5%
2.	Naa	18	45%
3.	Niram	0	0
4.	Mozhi	0	0
5.	Vizhi	0	0
6.	Malam	7	17.5%
7.	Moothiram	40	100%
8.	Naadi	40	100%



Observation

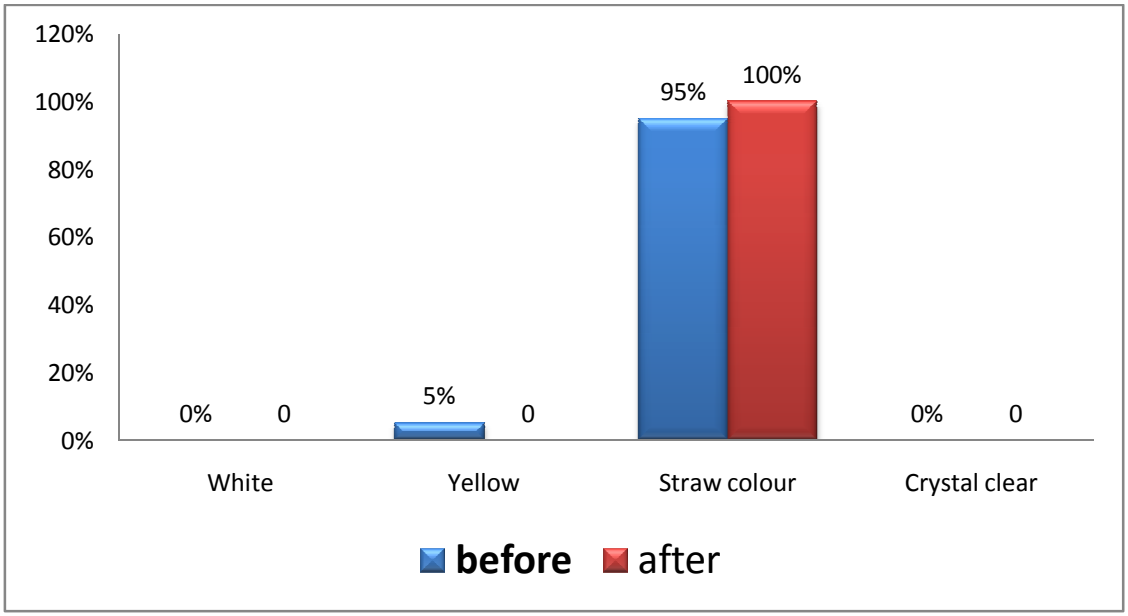
As per table 11a and Fig 11a: Naa was affected in 18 (45%) cases, Sparism was affected in 29 (72.5%) cases, Malam was affected in 7 (17.5%). Naadi and moothirakuri noted in all (100%) cases.

Neerkuri

Table-11b

Niram (colour):

S.No	Niram	Before Trt	After Trt
1.	White	0	0
2.	Yellowish	2(5%)	0
3.	Straw coloured	38 (95%)	40 (100%)
4.	Crystal clear	0	0
Total		40	40



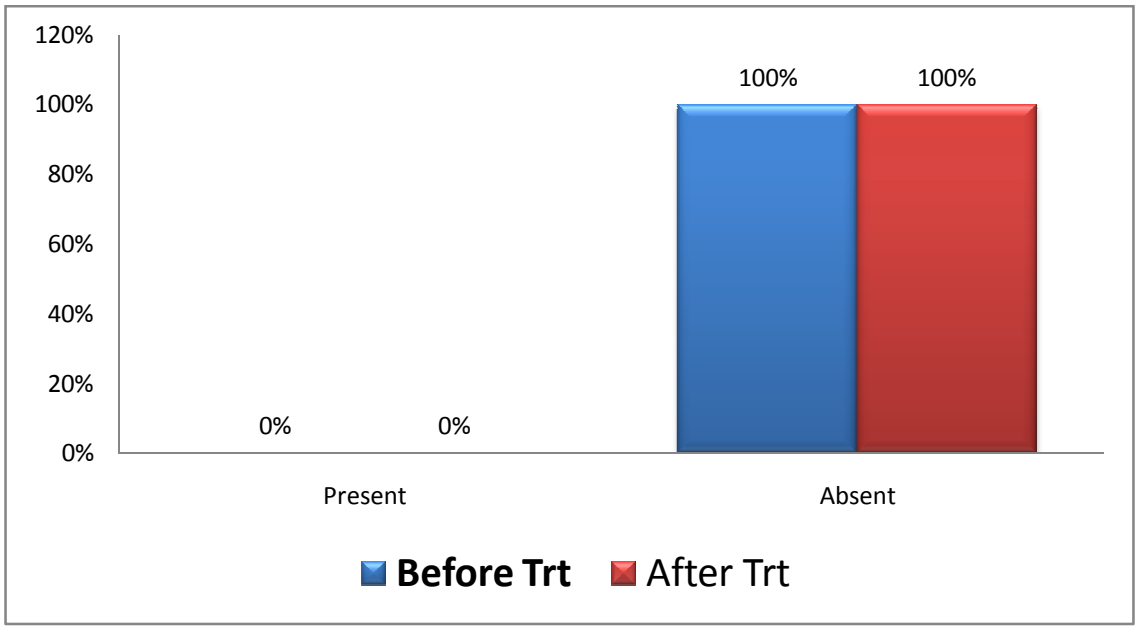
Observation:

As per table 11b and Fig 11b: Among the total of 40 patients before treatment straw coloured urine noted in 38 (95%) cases and yellow colour urine noted in 2 patients. In after treatment straw coloured noted in all (100%) cases.

Table-11c

Manam (Odour):

S.No	Manam	Before Trt	After Trt
1.	Present	0	0
2.	Absent	40 (100%)	40 (100%)
Total		40 (100%)	40 (100%)



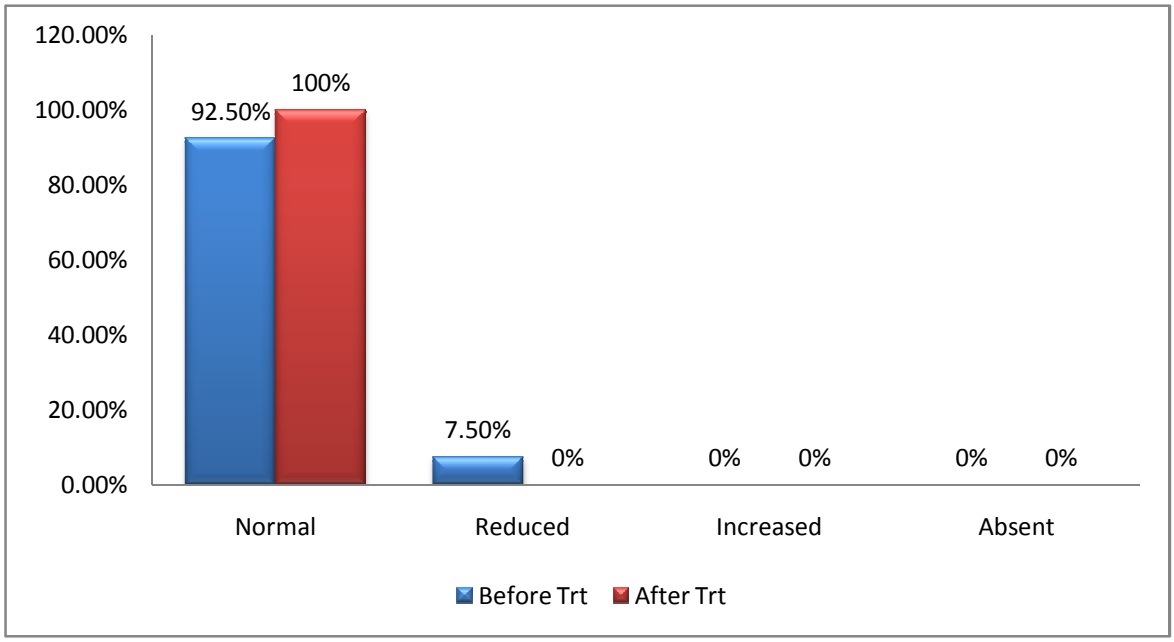
Observation:

As per table 11c and Fig 11c: Among the total of 40 patients Manam absent in all (100%) patients in before and after treatment.

Table-11d

Nurai (Froth):

S.No	Nurai	Before Trt	After Trt
1.	Normal	37 (92.5%)	40 (100%)
2.	Reduced	3 (7.5%)	0
3.	Increased	0	0
4.	Absent	0	0
Total		40	40



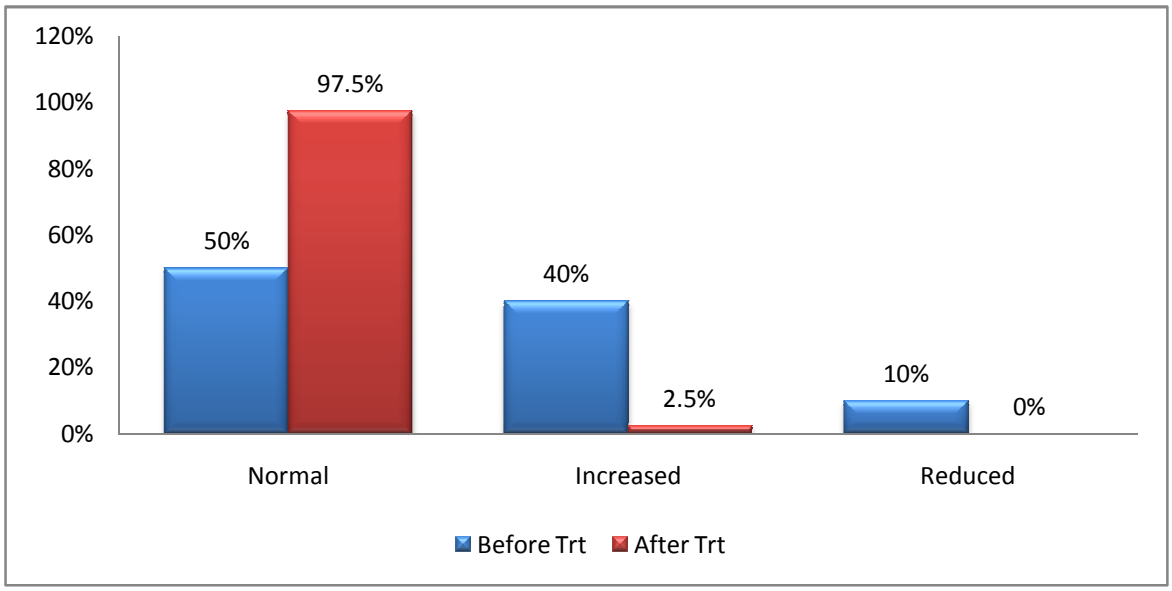
Observation:

As per table 11d and Fig 11d: Among the total of 40 patients before treatment Nurai normal in 37 (92.5%) cases and reduced in 3 (7.5%) cases. After treatment normal in all (100%) cases.

Table-11e

Edai (volume):

S.No	Edai	Before Trt	After Trt
1.	Normal	20 (50%)	39 (97.5%)
2.	Increased	16 (40%)	1 (2.5%)
3.	Reduced	4 (10%)	0
Total		40 (100%)	40 (100%)



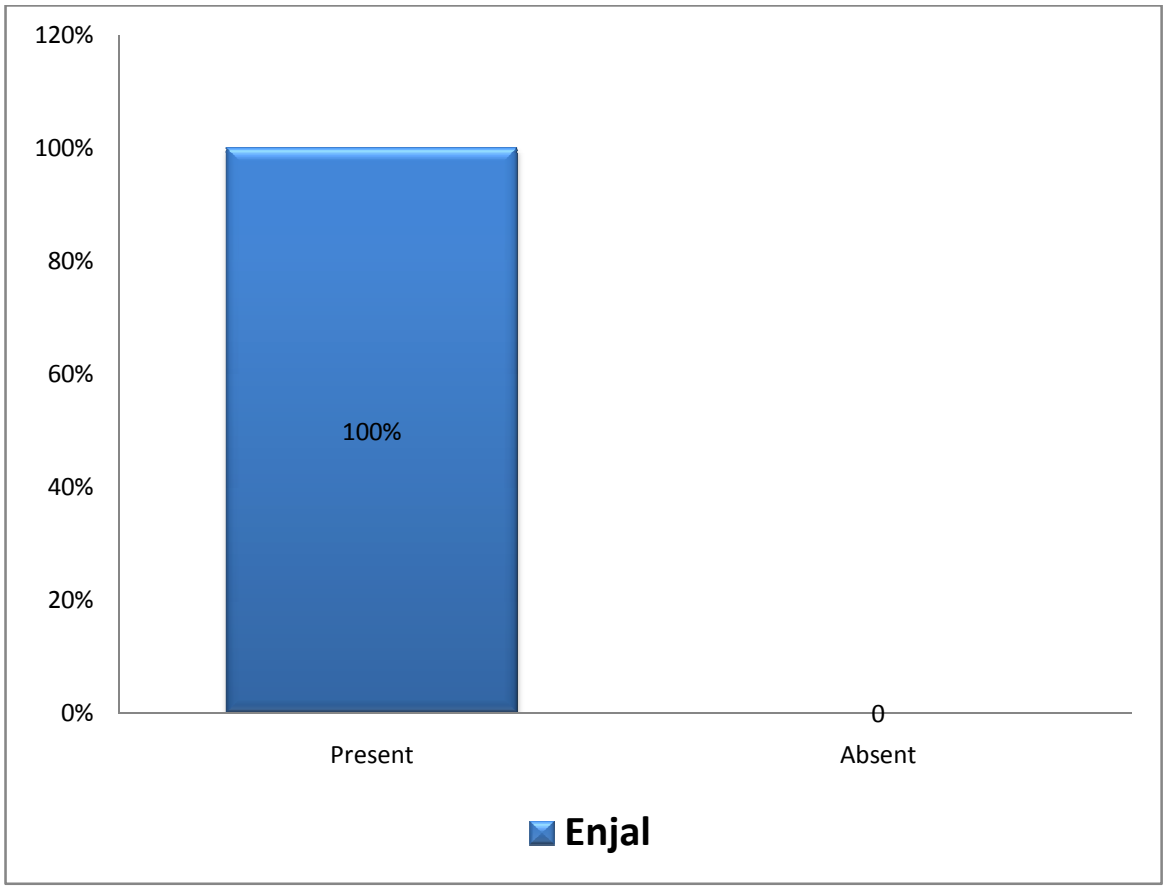
Observation:

As per table 11e and Fig 11e: Among the total of 40 patients before treatment Edai increased in 16 (40%) cases, reduced in 4 (10%) cases and normal in 20 (50%) cases. After treatment Edai normal in 39 (97.5%) cases and increased in 1 (2.5%) case.

Table-11f

Enjal (Deposits):

S.No	Enjal	No. of cases out of 40	Percentage (%)
1.	Present	40	100%
2.	Absent	0	0



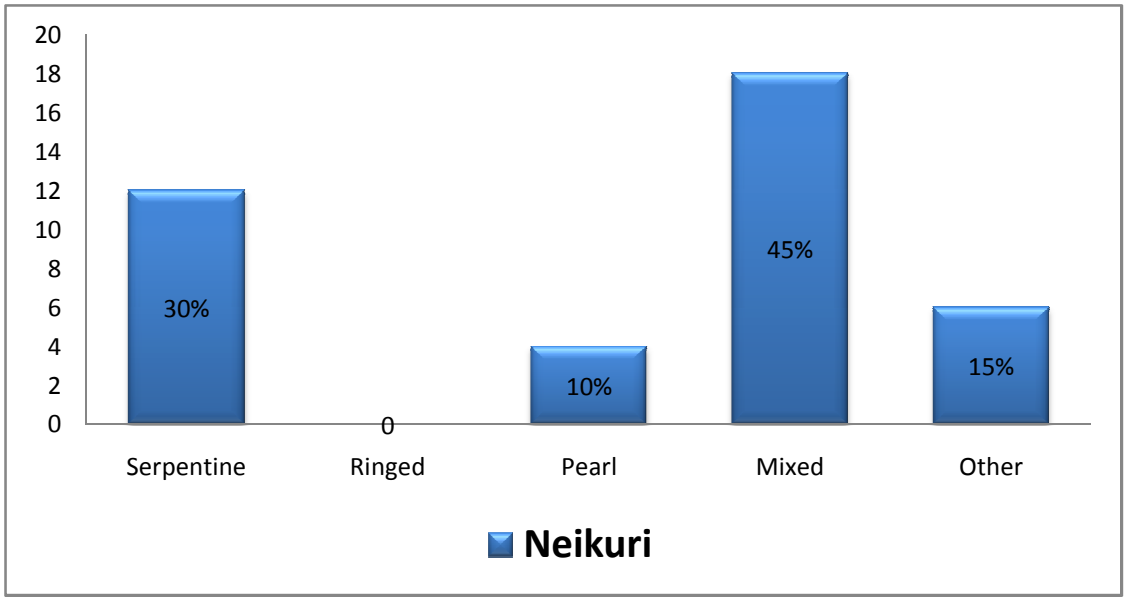
Observation:

As per table 11f and Fig 11f: Among the total of 40 patients Enjal noted in all (100%) patients' urine like pus cells and epithelial cells.

Table-11g

Neikuri:

S.No	Neikuri	No. of cases out of 40	Percentage (%)
1.	Serpentine like	12	30%
2.	Ring like	0	0
3.	Pearl like	4	10%
4.	Mixed pattern	18	45%
5.	Other pattern	6	15%



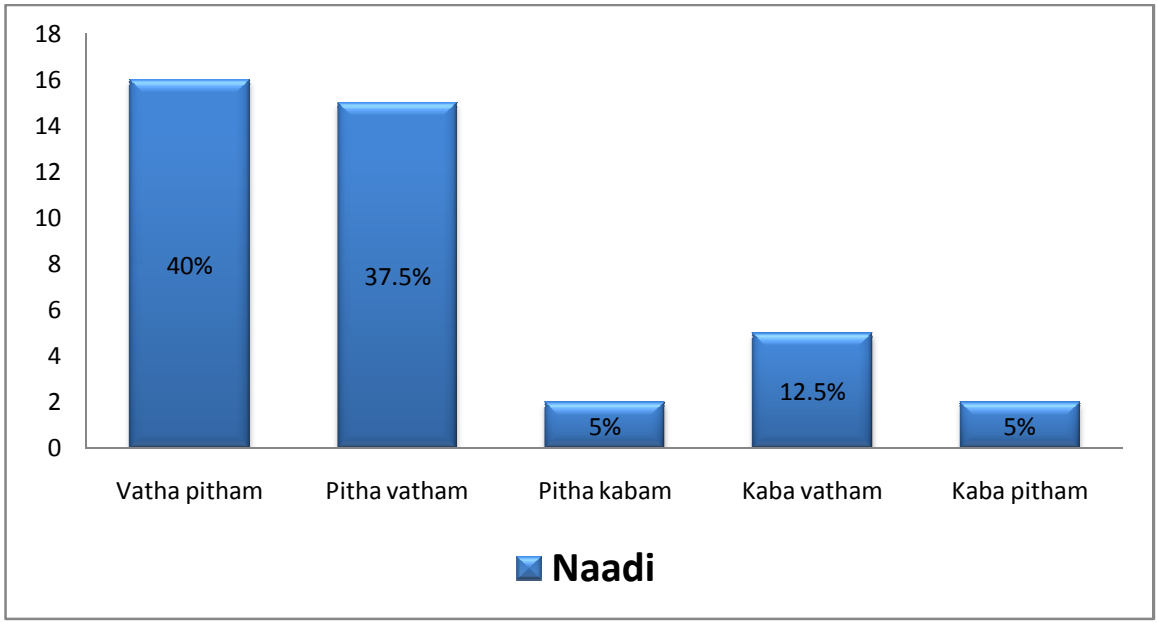
Inference:

As per table 11g and Fig 11g: Among the total of 40 patients Serpentine fashion is noted in 12 (30%) of cases, Pearl beaded fashion was noted in 4 (10%), Mixed fashion noted in 18 cases (45%) and other fashion noted in 6 cases (15%).

Table-11h

Naadi:

S.No	Naadi	No. of cases out of 40	Percentage (%)
1.	Vaatha piththam	16	40%
2.	Piththa vaatham	15	37.5%
3.	Piththa kapham	2	5%
4.	Kapha vatham	5	12.5%
5.	kappa pitham	2	5%



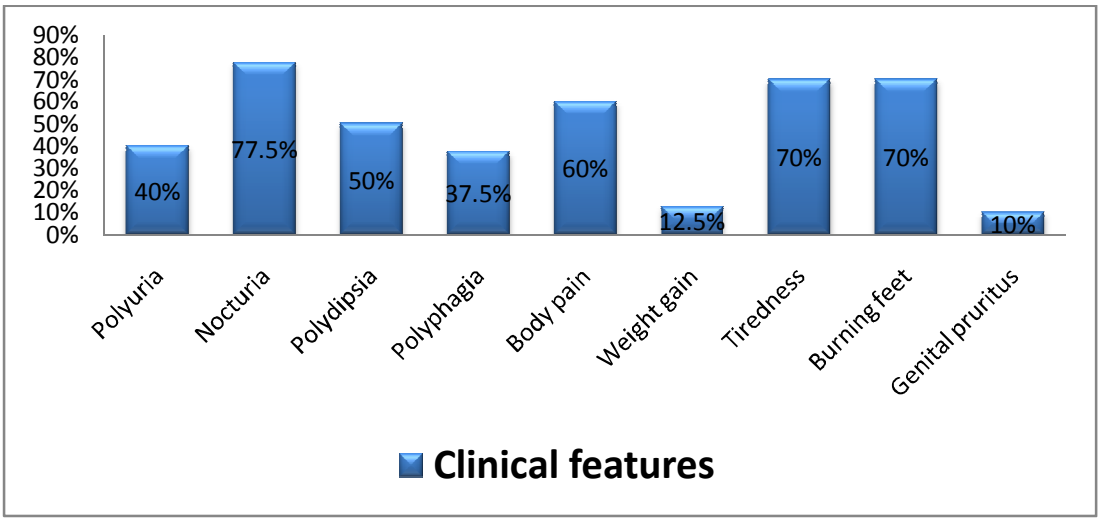
Inference:

As per table 11h and Fig 11h: Among 40 cases, 16 (40%) cases revealed Vatha pitha naadi and 15 (37.5%) cases with Pitha vatha naadi. Other 5 (12.5%) cases with Kaba vatham, 2 (5%) cases with Pitha kabam and 2 (5%) cases with Kaba pitham naadi.

Table-12

Showing the clinical features

S. no	Clinical features	No. of cases out of 40	Percentage (%)
1.	Polyuria	16	40%
2.	Nocturia	31	77.5%
3.	Polydipsia	20	50%
4.	Polyphagia	15	37.5%
5.	Body pain	24	60%
6.	Weight gain(obesity)	5	12.5%
7.	Tiredness	28	70%
8.	Burning feet	28	70%
9.	Genital pruritus	4	10%



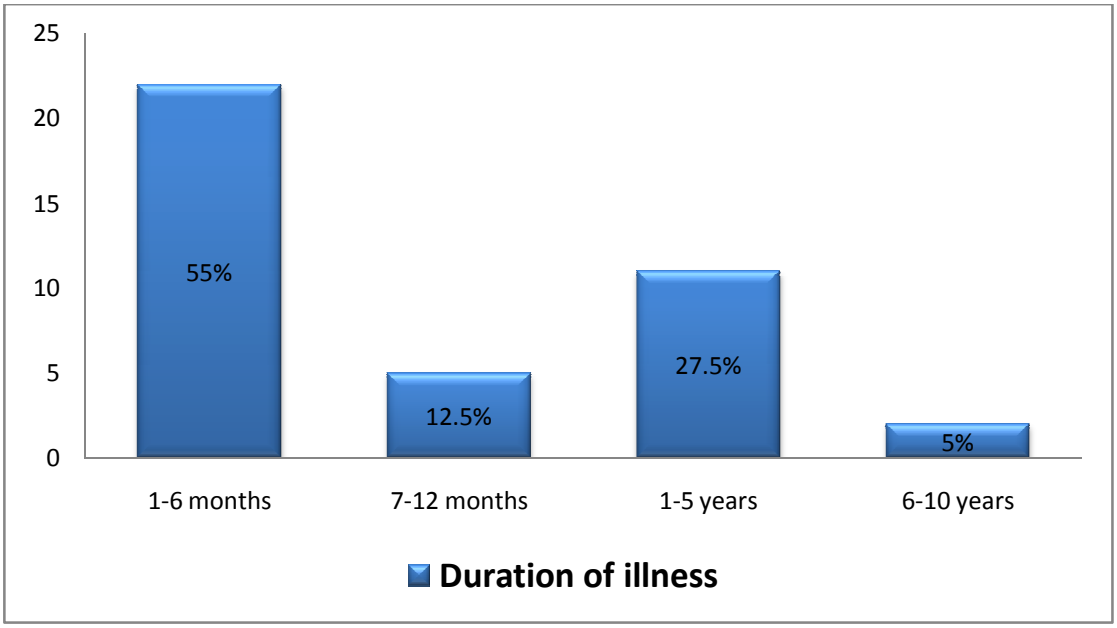
Inference:

As per table 12 and Fig 12: Among 40 cases, 16 cases (40%) patients complained of polyuria and 20 cases (50%) complained of Polydipsia. 15 cases (37.5%) complained of polyphagia, 31 cases (77.5%) complained nocturia, 28 cases (70%) complained tiredness, 24 cases (60%) complained body pain. 28 cases (70%) complained burning feet, and 4 cases (10%) complained genital pruritis, 5 cases (12.5%) complained weight gain.

Table-13

Chronicity of illness:

S.no	Duration of illness	No. of cases	Percentage (%)
1.	1-6 Months	22	55%
2.	7-12 Months	5	12.5%
3.	1-5 years	11	27.5%
4.	6-10 years	2	5%
	Total	40	100%



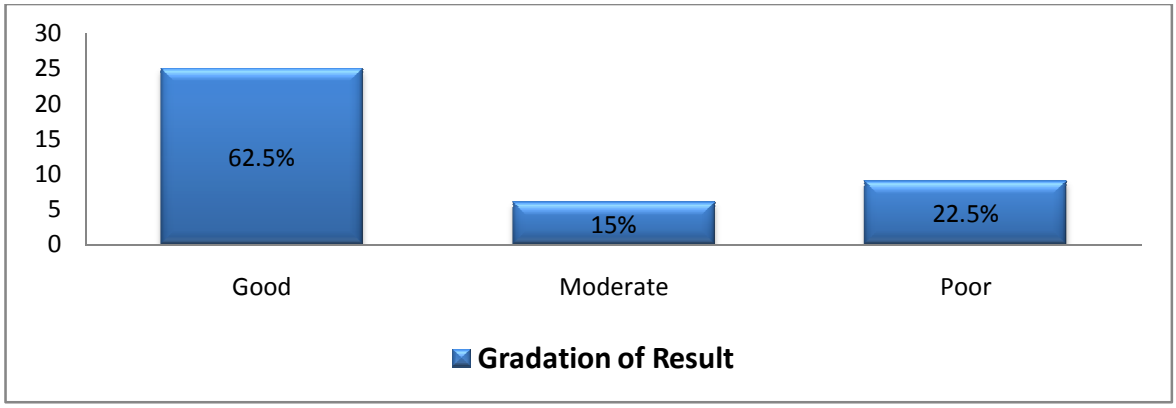
Inference:

As per table 13 and Fig 13: The chronicity of illness before recruitment for the study was more in 22 (55%) cases with 1-6 months of illness then 11 (27.5%) cases in the category with 1-5 years, 5 (12.5%) cases with 7-12 months and 2 (5%) cases with 6-10 years.

Table-14a **Gradation of results**

Blood sugar:

S.No	Gradation of results	No of cases	Percentage (%)
1.	Good	25	62.5%
2.	Moderate	6	15%
3.	Poor	9	22.5%
Total		40	100%



Inference: As per table 14 a,c,d,e and Fig 14a: Among 40 cases, 25 (62.5%) cases had Good clinical improvement, 6 (15%) cases had Moderate and 9 (22.5%) cases had Poor improvement.

Good result range: Fasting 70-126mg%, Postprandial-120-200mg% in 25 cases (62.5%)

Moderate result range: Fasting 127-140mg%, Postprandial-180-220mg% in 6 cases (15%)

Poor result range: Fasting above 140mg%, Postprandial- above 220mg% in 9cases (22.5%)

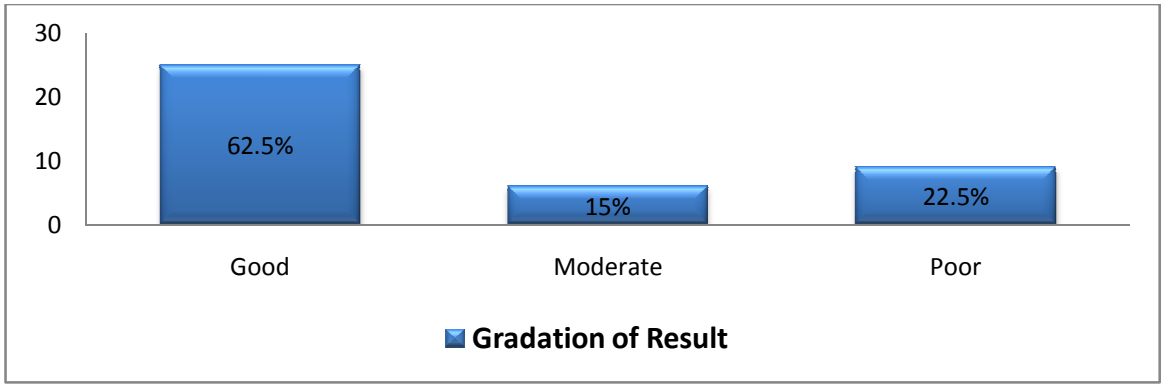
Note: Inclusion blood glucose range

- Fasting: 126 – 180mg%
- Postprandial: 200 – 300mg%

Table-14b **Gradation of results**

Urine sugar:

S.No	Gradation of results	No of cases	Percentage (%)
1.	Good	25	62.5%
2.	Moderate	6	15%
3.	Poor	9	22.5%
Total		40	100%



Inference: As per table 14 b,c,d,e and Fig 14b: Among 40 cases, 25 (62.5%) cases had Good clinical improvement, 6 (15%) cases had Moderate and 9 (22.5%) cases had Poor improvement.

Good result range: Fasting - Nil, Postprandial- Nil or + in 25 cases (62.5%)

Moderate result range: Fasting - Nil, Postprandial - + or Trace in 6 cases (15%)

Poor result range: Fasting – Nil or +, Postprandial- +, ++ in 9cases (22.5%)

Table-14c

Blood sugar and urine sugar before and after treatment results:

GOOD RESULTS										
S.No	**OP/IP No**	**Age/ Sex**	**Before treatment**		**After treatment**		**before treatment**		**after treatment**	
			B.Sugar mg/dl		**B.Sugar mg/dl**		**urine sugar**		**urine sugar**	
			FA	**PP**	**FA**	**PP**	**F**	**PP**	**F**	**PP**
1	C79506	52/F	135	210	115	161	Nil	++	Nil	Nil
2	C93762	48/F	140	235	118	170	Trace	++	Nil	+
3	C29305	50/F	160	230	125	154	Trace	++	Nil	Nil
4	C98043	40/F	138	210	121	174	Nil	+	Nil	Nil
5	C28386	50/F	131	212	120	196	Nil	+	Nil	+
6	C41076	44/F	176	278	121	198	Nil	++	Nil	+
7	C61757	45/F	131	266	122	161	Nil	N	Nil	Nil
8	4244	45/F	126	219	115	170	Nil	++	Nil	Nil
9	4151	55/F	142	272	126	152	Nil	N	Nil	Nil
10	4182	53/F	156	252	112	167	Nil	+	Nil	Nil
11	4210	40/F	127	226	112	123	Nil	N	Nil	Nil
12	4203	55/F	127	210	122	136	Nil	N	Nil	Nil
13	4129	48/F	138	261	110	135	N	N	Nil	Nil
14	4236	55/F	134	300	107	191	+	++	Nil	Nil
15	C77100	50/M	136	239	126	146	Nil	+	Nil	Nil
16	C93323	40/M	136	254	112	164	Nil	+	Nil	Nil
17	C77872	33/M	117	257	110	190	Trace	+	Nil	+
18	C76014	43/M	157	245	120	170	Nil	+	Nil	Trace
19	C73821	43/M	124	200	118	177	Nil	N	Nil	Nil
20	C77322	53/M	134	236	121	177	Nil	++	Nil	Nil
21	C84379	40/M	173	265	123	195	Trace	++	Nil	+
22	C75253	36/M	128	237	116	199	Nil	Trace	Nil	+
23	B95665	42/M	145	213	122	164	N	+	Nil	Nil
24	C92670	46/M	160	270	126	177	+	++	Nil	Nil
25	C99837	41/M	128	268	112	187	N	+	Nil	+

Table-14d

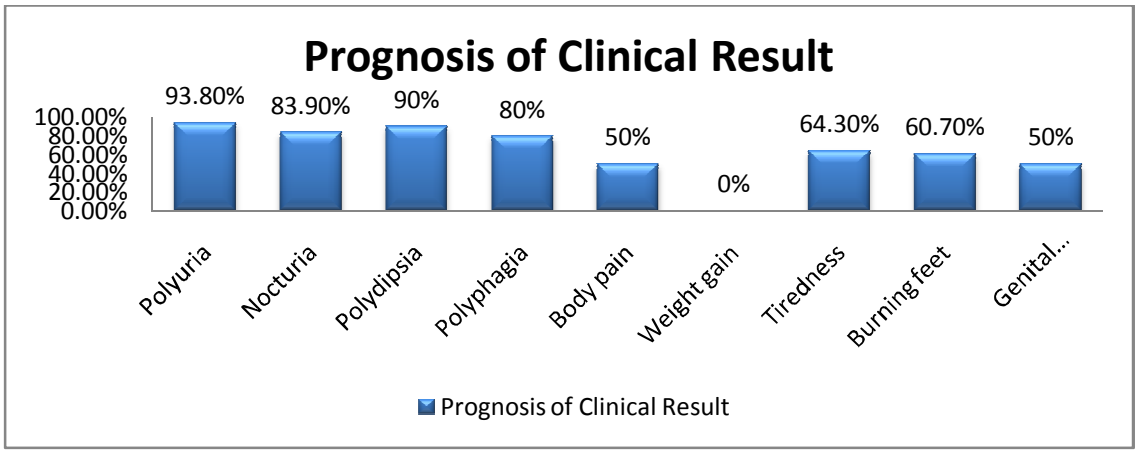
MODERATE RESULTS										
S.No	OP/IP No	Age/ Sex	Before treatment		After treatment		before treatment		after treatment	
			B.Sugar mg/dl		B.Sugar mg/dl		urine sugar		urine sugar	
			FA	PP	FA	PP	F	PP	F	PP
1	4272	54/F	138	233	130	192	+	++	Nil	+
2	C81031	53/M	172	260	140	211	Trace	+	Nil	+
3	C92864	38/M	162	258	139	198	Nil	+	Nil	+
4	C42893	43/M	131	278	130	196	Nil	++	Nil	+
5	C85416	42/M	160	220	139	180	Nil	+	Nil	Trace
6	C89234	50/M	159	298	140	182	Trace	+	Nil	Trace

Table-14e

POOR RESULTS										
S.No	OP/IP No	Age/ Sex	Before treatment		After treatment		before treatment		after treatment	
			B.Sugar mg/dl		B.Sugar mg/dl		urine sugar		urine sugar	
			FA	PP	FA	PP	F	PP	F	PP
1	4125	50/F	191	234	160	174	+	++	Nil	+
2	4035	52/F	169	239	185	336	Nil	++	Nil	++
3	B53440	42/F	168	295	143	225	Nil	++	Nil	++
4	C77041	51/M	132	262	154	220	Nil	++	Nil	++
5	C91330	53/M	148	300	148	220	Nil	+	Nil	++
6	C86489	55/M	146	278	156	280	Nil	+	Nil	++
7	B95283	50/M	132	300	149	248	Nil	+	Nil	++
8	C78746	42/M	160	290	252	300	Nil	++	+	++
9	C90731	45/M	180	295	147	191	+	++	Trace	++

Table-15 Prognosis of Clinical features results:

Clinical features		Prognosis Result	
	Before treatment	After treatment	Percentage
Polyuria	16	15	93.8%
Nocturia	31	26	83.9%
Polydipsia	20	18	90%
Polyphagia	15	12	80%
Body pain	24	12	50%
Weight gain (obesity)	5	0	0%
Tiredness	28	18	64.3%
Burning feet	28	17	60.7%
Genital pruritus	4	2	50%



Inference:

As per table 15 and Fig 15: After treatment there was a considerable reduction in all symptoms except obesity. Good reduction in Polyuria (93.8%), Polydipsia (90%), Polyphagia (80%) and Nocturia (83.9%), Moderate reduction in Tiredness (64.3%) and burning feet (60.7%) and then poor reduction in Genital Pruritis (50%) and Body pain (50%).

Statistical Analysis

All collected data were entered into MS Excel software using different columns as variables and rows as patients. SPSS software was used to perform statistical analysis. Basic descriptive statistics include frequency distributions and cross-tabulations were performed. The quantity variables were expressed as Mean ± Standard Deviation and qualitative data as percentage. A probability value of <0.05 was considered to indicate as statistical significance. Paired't' test was performed for determining the significance between before and after treatment.

Blood Glucose at fasting	Mean ± Std	t value and p value
Before trt.	146.18 ± 18.22	T = 3.81, p < 0.0001
After trt	131.6 ± 25.7	

The mean ± standard deviation of glucose at fasting before and after treatment were 146.18 ± 18.22 and 131.6 ± 25.7 respectively which is statistically significant (t= 3.81 p<0.0001).

Blood Glucose at Postprandial	Mean ± Std	t value and p value
Before trt.	250.00 ± 33.965	T = 9.044, p < 0.0001
After trt	191.70 ± 43.439	

The mean ± standard deviation of glucose at postprandial before and after treatment were 250.00 ± 33.965and 191.70 ± 43.439 respectively which is statistically significant (T = 9.044, p < 0.0001).

Serum total cholesterol	Mean ± Std	t value and p value
Before trt.	195.83 ± 43.974	T = 3.572, p < 0.001
After trt	170.98 ± 27.647	

The mean ± standard deviation of serum total cholesterol before and after treatment were 195.83 ± 43.974 and 170.98 ± 27.647respectively which is statistically significant (T = 3.572, p < 0.001).

HDL	Mean ± Std	t value and p value
Before trt.	38.70 ± 8.064	T = 2.666, p < 0.011
After trt	35.70 ± 4.968	

The mean ± standard deviation of HDL before and after treatment were 38.70 ± 8.064 and 35.70 ± 4.968 respectively which is statistically significant (T = 2.666, p < 0.011).

LDL	Mean ± Std	t value and p value
Before trt.	108.80 ± 28.776	T = 3.799, p < 0.0001
After trt	93.40 ± 16.375	

The mean ± standard deviation of LDL before and after treatment were 108.80 ± 28.776 and 93.40 ± 16.375 respectively which is statistically significant (t= 3.799 p<0.0001).

VLDL	Mean ± Std	t value and p value
Before trt.	41.93 ± 15.698	T = 3.479, p < 0.001
After trt	33.78 ± 11.939	

The mean ± standard deviation of VLDL before and after treatment were 41.93 ± 15.698 and 33.78 ± 11.939 respectively which is statistically significant (t= 3.479 p<0.001).

TGL	Mean ± Std	t value and p value
Before trt.	198.95 ± 81.305	T = 3.272, p < 0.002
After trt	159.05 ± 51.591	

The mean ± standard deviation of TGL before and after treatment were 198.95 ± 81.305 and 159.05 ± 51.591 respectively which is statistically significant (t= 3.272 p<0.002).

DISCUSSION

Diabetes Mellitus is a heterogeneous group of metabolic disorders characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. Type 2 Diabetes is one of the major health problems all over the world.

Mathumegam mentioned in siddha literature may be correlated to "Diabetes mellitus" having the symptoms of polyuria (frequent urination), polydipsia (increased thirst), polyphagia (increased hunger). *Yoogimuni,* author of *Yoogi Vaithiya Chinthamani* has described the signs and symptoms of *Mathumegam* as "Gunam and *Avathaigal"*. They are also may be correlated to sign and symptoms of Diabetes mellitus.

In the siddha literature *Agathiyar 2000*- 3rd volume, the drug ***Atthippattaiyathi kasayam*** is indicated for ***Mathumegam*** (type-2 Diabetes Mellitus). *Atthippattaiyathi kasayam* comprises of twenty two Herbs. There are 12 herbs of this formulation having potential Antidiabetic activity and 12 herbs have potential Antioxidant activity and other herbs have Hypolipidemic, Hepatoprotective, Cardioprotective, Nephroprotective action. So the formulation is may be a preventable medicine for development of diabetes and its complications.

The raw drugs were purchased from reputed country drugs stores and authenticated by the concerned department. The trial drug was prepared by the standard operating procedure as mentioned in the protocol in the Dept of Gunapadam, National Institute of Siddha under the direct supervision of lecturers.

The safety of the trial drug usage and standardization of the trial drug through biochemical analysis were also ensured during the study.

The preclinical toxicity studies (Acute and long term toxicity) for the above said trial drug was carried out at National Institute of Siddha after getting the proper acceptance and permission from the Institutional Animal Ethical Committee. The trial drug was proved to be safe for human beings from the observations made from the study.

The biochemical qualitative and quantitative analysis were done at the biochemistry lab of NIS and SAIF, IIT Chennai respectively. It revealed the presence of effective minerals and the existence of the drug molecules at micro level.

Before initiating the trial, Institutional Animal Ethical Committee (IAEC: 1248/ac/09/ CPCSEA/4-07/2011 - Dt. 20.12.2011) and Institutional Ethical Committee (IEC: NIS/IEC/ 2011/3/07 - Dt. 24.12.2011) of NIS, approval was obtained for this study.

After the approval of the Institutional Ethical Committee of NIS, the clinical study was conducted with a well defined protocol with a proper proforma under the direct supervision of faculties of Dept of Maruthuvam. After screening 60 cases reporting at the OPD, 40 cases were selected for induction to the trial. The patient who satisfied the Inclusion and Exclusion criteria as per the protocol were admitted to the trial. The patients were well explained about the clinical trial and informed consent was obtained from the patient.

Then the enrolled patients were subjected to lab investigations before and after treatment. From the 1stday onwards the patients were treated with the trial drug ***Atthippattaiyathi kasayam*** 336ml internal 3 times daily for a period of 40 days.

For OP patients, they should visit the hospital once in 10 days. At each clinical visit clinical assessment was done and prognosis was noted. For IP Patients clinical assessment was done daily and prognosis was noted.

For IP patients who were not in a situation to stay in the hospital for a long time were advised to attend the OPD for the continuation of the treatment. All the patients were put under observation for 2 months as follow up period without the trial drug treatment. The observations are summarized below.

Incidence with reference to Age distribution:

The prevalence of the disease was found to be higher in 18 cases (45 %) in the age group of 51 - 55 years, 13 cases (32.5%) in the age group of 41 – 45 years, 6 cases (15 %) in the age group of 46 - 50 years and 3 cases (7.5 %) in the age group of 30 - 40 years.

Incidence with reference to Sex distribution:

Among the 40 patients selected, prevalence of the disease was found to be higher in males i.e. 22 cases (55%) then the Female cases of 18 (45%).

Incidence with reference to Socio-economic status:

The incidence of the disease was found to be higher in 20 (50 %) cases belonging to poor class, medium in 14 (35 %) cases belonging to middle class and lower in 6 (15) cases belonging to high class. Affected cases were mostly from poor and middle class.

Incidence with reference to Family history:

Among the 40 cases, Positive familial history was seen in 24 (60 %) patients and no history of family involvement was found in 16 cases(40 %). From the above study hereditary plays an important role in this disease.

Incidence with reference to Food habits:

Among 40 cases the incidence of the disease was higher in 33 (82.5%) cases, which were non vegetarians and lower in vegetarians 7 cases (17.5%). It clearly showed that non-vegetarians is more prone to Mathumegam.

Incidence with reference to Thinai:

Among 40 cases 31 (77.5%) cases belongs to Neidhal, 7 (17.5%) cases were from Marutham, and only one (2.5%) case was from Kurunji and Mullai.

Incidence with reference to Paruva kaalangal:

Among the 40 cases, in 23 cases (57.5%) the incidence of the disease seems to be higher in Kaar kaalam (Avani-Puratasi), 17 cases (42.5%) in Muthuvenil kaalam (Aani-Aadi).

Incidence with reference to Yakkai:

Among the 40 cases, 12 cases (30%) were vaatha thegi, 7cases (17%) were pitha thegi, 11 cases (28%) were kappa thegi and 10 cases (25%) were thontha thegi.

Incidence with reference to Vatham:

Among 40 cases, Samanan and viyanan were affected in 28 (70%) cases. In 34 (85 %) cases Devathathan was affected. Abanan was affected in 23 (57.5%) cases and Udhanan was affected in 2 (5%) cases.

Incidence with reference to Pitham:

Among the 40 cases, Sathagam was affected in 31 (77.5%) cases and Anarpitham was affected in 14 (35%) cases.

Incidence with reference to Kabham:

Among 40 cases Avalambagam was affected in 2 (5%) cases and Santhigam was affected in 14 (35 %) as a result of pain in both lower limb joints.

Incidence with reference to Ezhu udal thathukkal:

A mong 40 cases, Saaram was affected in all the 40 (100%) cases, Senner was affected in 5 (12.5 %) cases, Kozhuppu affected in 17 cases (42.5%) and Enbu affected in 10 cases (25%).

Incidence with reference to Envagai thervugal:

In enn vagai thervugal Naa was affected in 18 (45%) cases, Sparism was affected in 29 (72.5%) cases, Malam was affected in 7 (17.5%). Naadi and moothirakuri noted in all (100%) cases.

Neerkuri:

- **Niram:**
    - Among the total of 40 patients before treatment straw coloured urine noted in 38 (95%) cases and yellow colour urine noted in 2 patients. In after treatment straw coloured noted in all (100%) cases.
- **Manam:**
    - Among the total of 40 patients Manam absent in all (100%) patients in before and after treatment.
- **Nurai:**
    - Among the total of 40 patients before treatment Nurai normal in 37 (92.5%) cases and reduced in 3 (7.5%) cases. After treatment normal in all (100%) cases.
- **Edai (volume):**
    - Among the total of 40 patients before treatment Edai increased in 16 (40%) cases, reduced in 4 (10%) cases and normal in 20 (50%) cases. After treatment Edai normal in 39 (97.5%) cases and increased in 1 (2.5%) case.
- **Enjal:**
    - Among the total of 40 patients Enjal noted in all (100%) patients' urine like pus cells and epithelial cells.

Neikuri:

Among the total of 40 patients Serpentine pattern is noted in 12 (30%) of cases, Pearl pattern was noted in 4 (10%), Mixed pattern noted in 18 cases (45%) and other pattern noted in 6 cases (15%).

Naadi:

Among 40 cases, 16 (40%) cases revealed Vatha pitha naadi and 15 (37.5%) cases with Pitha vatha naadi. Other 5 (12.5%) cases with Kaba vatham, 2 (5%) cases with Pitha kabam and 2 (5%) cases with Kaba pitham naadi.

Incidence with reference to clinical features:

16 cases (40%) patients complained of polyuria and 20 cases (50%) complained of Polydipsia. 15 cases (37.5%) complained of polyphagia, 31 cases (77.5%) complained nocturia, 28 cases (70%) complained tiredness, 24 cases (60%) complained body pain. 28 cases (70%) complained burning feet, and 4 cases (10%) complained genital pruritis, 5 cases (12.5%) complained weight gain.

Chronicity of illness:

The chronicity of illness before recruitment for the study was more in 22 (55%) cases with 1-6 months of illness then 11 (27.5%) cases in the category with 1-5 years, 5 (12.5%) cases with 7-12 months and 2 (5%) cases with 6-10 years.

Gradation of results:

Blood Glucose:

The result obtained from the clinical case study, 25 (62.5%) cases had Good clinical improvement, 6 (15%) cases had Moderate and 9 (22.5%) cases had Poor improvement.

Good result range:

Fasting 70-126mg%, Postprandial-120-200mg% in 25 cases (62.5%)

Moderate result range:

Fasting 127-140mg%, Postprandial-180-220mg% in 6 cases (15%)

Poor result range:

Fasting above 140mg%, Postprandial- above 220mg% in 9cases (22.5%)

Note: Inclusion blood glucose range

- Fasting: 126 – 180mg%
- Postprandial: 200 – 300mg%

Urine Glucose:

The result obtained from the clinical case study, 25 (62.5%) cases had Good clinical improvement, 6 (15%) cases had Moderate and 9 (22.5%) cases had Poor improvement.

Good result range: Fasting-Nil, Postprandial- Nil or + in 25 cases (62.5%)

Moderate result range: Fasting-Nil, Postprandial - + or Trace in 6 cases (15%)

Poor result range: Fasting-Nil or +, Postprandial- +, ++ in 9cases (22.5%)

Prognosis of Clinical features:

After treatment there was a considerable reduction in all symptoms except obesity. Good reduction in Polyuria (93.8%), Polydipsia (90%), Polyphagia (80%) and Nocturia (83.9%), Moderate reduction in Tiredness (64.3%) and burning feet (60.7%) and then poor reduction in Genital Pruritis (50%) and Body pain (50%).

Statistical Analysis:

With the evidence of statistical report, it shows the average fasting blood sugar before the treatment and after the treatment were 146 and 131 respectively, postprandial blood sugar respectively 250 and 191. There is a significant difference between before and after treatment in the level of blood sugar at Fasting and Postprandial [$P < 0.0001$].

The mean ± standard deviation of serum total cholesterol before and after treatment were 195.83 ± 43.974 and 170.98 ± 27.647respectively which is statistically significant ($T = 3.572$, $p < 0.001$).

SUMMARY

- The aim of the study was to evaluate the safty and efficacy of the drug **Atthippattaiyathi kasayam** in the treatment of Mathumegam.
- Before initiating the clinical trial, approval was got from the Institutional Animal Ethical Committee (IAEC: 1248/ac/09/CPCSEA/4-07/2011 - Dt. 20.12.2011) and Institutional Ethical Committee (IEC: NIS/IEC/2011/3/07 - Dt. 24.12.2011) for conducting the pre clinical studies and clinical studies respectively by submitting the well defined protocol and proforma.
- The raw drugs were authenticated by the concerned department and the trial drug was prepared in the Gunapadam lab of National Institute of Siddha as per the Standard Operating Procedure mentioned in the protocol.
- The medicine was then subjected to toxicity studies (Acute and long term toxicity studies) as per the protocol and the safety of the drug was ensured.
- The qualitative and quantitative bio chemical studies were done at the bio chemistry lab of National Institute of Siddha.
- SEM analysis and trace metals detection were done at Sophisticated Analytical Instrument Facility, IIT, Chennai.
- The Biochemical analysis of the trial drug was done in Biochemistry laboratory National Institute of Siddha. Biochemical analysis showed the presence of inevitable constituents like Calcium, Iron, Sulphate.
- Oral toxicity studies conducted ensured the safety usage of the drug to animals up to a maximum dose of 270 mg/animal for mice and 2700 mg/animal for wister rat.
- Histopathological studies was done in pathology lab, Vels University, it shows that there is no abnormalities in the studies.
- Among the 60 cases screened at the OPD of Branch-I, Ayothidoss Pandithar Hospital, NIS, 40 cases were recruited for the trial as per the inclusion and exclusion criteria of the Protocol.
- Clinical diagnosis of Mathumegam was made by Siddha and Modern methodology.
- Before inducement into the trial informed consent was obtained from the patients. Out of the 40 cases 30 cases were treated in OPD and 10 cases in IPD in Branch-I

- The clinical trial was conducted with the trial medicine Atthippattaiyathi kasayam 5g t.i.d for a period of 40 days, referred under the Siddha literature *Yoogi Vaithiya Chinthamani.*
- Diet restriction was strictly followed during the period of drug administration as per noted in the form IV D (Dietary advice form).
- Required lab investigations were carried out before and after the treatment and the concerned data was recorded in the proforma.
- Clinical assessment was done daily in all the IP patients and in OP patients it was assessed once in 10 days.
- During the study period, there was no event of any adverse reactions owing to the drug or disease.
- The clinical and labarotary Obserevation in the Mathumegam patient treated with the trial drug **Atthippattaiyathi kasayam** showed significant control in their blood glucose level and upgrading with in general health.
- Statistical analysis showed significant difference between before and after treatment in the level of blood sugar at Fasting and Postprandial [$P < 0.0001$].
- In these studies out of 40 cases, 25 cases (62.5%) had good clinical improvement, 6 cases (15%) had moderate and 9 cases (22.5%) had poor improvement.

CONCLUSION

- The safety studies (the acute toxicity and long term toxicity) studies conducted revealed that the trial drug was safe even at higher dosage of 270 mg/animal for mice and 2700 mg/animal for wister rat. There were no abnormalities found in histopathological examination. Hence it can be reasonably assumed that the drug is safe for human.
- Clinical study revealed that the trial drug posses' good clinical improvement in 25 (62.5%) cases, 6 (15%) cases had moderate results and 9 (22.5%) cases had poor results.
- The clinical and labarotary Obserevation in the Mathumegam patient treated with the trial drug Atthippattaiyathi kasayam showed significant control in their blood glucose level and upgrading with in general health.
- It is undoubtedly marked that the Medicine used in the siddha system do not have any harmful side effects.
- Because of the hopeful results clinically, the study may be undertaken with the same drug for a prolonged period of time in more number of cases and it may throw new lights in the management of Mathumegam.

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BIO -CHEMICAL ANALYSIS OF ATTHIPPATTAIYATHI KASAYAM - ANALYSED AT NATIONAL INSTITUTE OF SIDDHA

S.No	EXPERIMENT	OBSERVATION	INFERENCE
1.	Appearance of sample	Brown in colour	
2.	**Solubility:** a.A little(500mg) of the sample is shaken well with distilled water. b.A little(500mg) of the sample is shaken well with con. HCl/Con. H_2So_4	Sparingly soluble	Presence of Silicate
3.	**Action of Heat:** A small amount(500mg) of the sample is taken in a dry test tube and heated gently at first and then strong.	White fumes evolved	Presence of Carbonate
4.	**Flame Test:** A small amount(500mg) of the sample is made into a paste with con. HCl in a watch glass and introduced into non-luminous part of the Bunsen flame.	No Bluish green flame appeared.	Absence of Copper
5.	**Ash Test:** A filter paper is soaked into a mixture of sample and dil. cobalt nitrate solution and introduced into the Bunsen flame and ignited.	Yellow colour flame	Presence of sodium

Preparation of Extract

5 gm of Atthippattaiyathi Kasayam powder is weighed accurately and placed in a 250 ml clean beaker and added with 50 ml of distilled water. Then it is boiled well for about 10 minutes. Then it is cooled and filtered in a 100ml volumetric flask and made up to 100ml with distilled water.

S.No	EXPERIMENT	OBSERVATION	INFERENCE
	I. Test For Acid Radicals		
1.	**Test For Sulphate:** a.2ml of the above prepared extract is taken in a test tube to this added 2ml of 4% dil ammonium oxalate solution.	Cloudy appearance present	Presence of Sulphate
2.	**Test For Chloride:** 2ml of the above prepared extracts is added with 2ml of dil-HCl is added until the effervescence ceases off..	No cloudy appearance present	Absence of Chloride
3.	**Test For Phosphate:** 2ml of the extract is treated with 2ml of dil.ammonium molybdate solution and 2ml of con.HNo3	Mild Yellow appearance present	Presence of Phosphate
4.	**Test For Carbonate:** 2ml of the extract is treated with 2mldil. magnesium sulphate solution	Cloudy appearance present	Absence of carbonate
C	**Test For Nitrate:** 1gm of the substance is heated with copper turning and concentrated H2So4 and viewed the test tube vertically down.	No Brown gas is evolved	Absence of Nitrate
6.	**Test For Sulphide:** 1gm of the substance is treated with 2ml of con. HCL	NoRotten Egg Smelling eggs evolved	Absence of Sulphide
7.	**Test For Fluoride & Oxalate:** 2ml of extract is added with 2ml of dil.	No Cloudy appearance	Absence of fluoride and oxalate

	Acetic acid and 2ml dil.calcium chloride solution and heated**.**		
8.	**Test For Nitrite:** 3drops of the extract is placed on a filter paper, on that-2 drops of dil.acetic acid and 2 drops of dil.Benzidine solution is placed.	No Characteristic changes	Absence of Nitrite
9.	**Test For Borate:** 2 Pinches(50mg) of the substance is made into paste by using dil.sulphuric acid and alcohol (95%) and introduced into the blue flame.	Bluish green colour flame not appeared	Absence of borate
	II. Test For Basic Radicals		
1.	**Test For Lead:** 2ml of the extract is added with 2ml of dil.potassium iodine solution.	NoYellow Precipitate is obtained.	Absence of Lead
2.	**Test For Copper:** **a.**One pinch(50mg) of substance is made into paste with con. HClin a watch glass and introduced into the non-luminuous part of the flame.	No Blue colour flame No Blue colour precipitate formed.	Absence of copper
3.	**Test For Aluminium:** To the 2ml of extract dil.sodium hydroxide is added in 5 drops to excess.	Yellow colour appeared	Presence of aluminium
4.	**Test For Iron:** a.To the 2ml of extract add 2ml of dil.ammonium solution b.To the 2ml of extract 2ml thiocyanate solution and 2ml of con HNo3 is added	mild red colour appear	Presence of Iron
5.	**Test For Zinc:** To 2ml of the extract dil.sodium hydroxide solution is added in 5 drops to excess and dil.ammonium chloride is added.	White precipitate is not formed	Absence of Zinc
6.	**Test For Calcium:**		

	2ml of the extract is added with 2ml of 4% dil.ammonium oxalate solution	Cloudy appearance and white precipitate is obtained	Presence of calcium
7.	**Test For Magnesium:** To 2ml of extract dil.sodium hydroxide solution is added in drops to excess.	White precipitate is obtained	Absence of Magnesium
8.	**Test For Ammonium:** To 2ml of extract 1 ml of Nessler's reagent and excess of dil.sodium hydroxide solution are added.	Brown colour appeared	Presence of ammonium
9.	**Test For Potassium**: A pinch(25mg) of substance is treated of with 2ml of dil.sodium nitrite solution and then treated with 2ml of dil.cobalt nitrate in 30% dil.glacial acetic acid	No Yellowish precipitate is obtained.	Absence of Potassium
10.	**Test For Sodium**: 2 pinches(50mg) of the substance is made into paste by using HCl and introduced into the blue flame of Bunsen burner.	yellow colour flame appeared	Presence of sodium
11.	**Test For Mercury**: 2ml of the extract is treated with 2ml of dil.sodium hydroxide solution.	No yellow precipitate is obtained	Absence of mercury
12.	**Test For Arsenic:** 2ml of the extract is treated with 2ml of dil.sodium hydroxide solution.	No brownish red precipitate is obtained	Absence of arsenic
	III. Miscellaneous		
1.	**Test For Starch**: 2ml of extract is treated with weak dil iodine solution	Red colour developed	Absence of starch
2.	**Test For Reducing Sugar:**		Presence of reducing

	5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 8 to 10 drops of the extract and again boil it for 2 minutes. The colour changes are noted.	Brick red colour not developed	sugar
3.	**Test For The Alkaloids**: a) 2ml of the extract is treated with 2ml of dil.potassium lodide solution. b) 2ml of the extract is treated with 2ml of dil.picric acid. c) 2ml of the extract is treated with 2ml of dil.phosphotungstic acid.	. Yellow colour developed	Presence of Alkaloid
4.	**Test For Tannic Acid:** 2ml of extract is treated with 2ml of dil.ferric chloride solution	No block precipitate is obtained	Presence of Tannic acid
5.	**Test For Unsaturated Compound**: To the 2ml of extract 2ml of dil.Potassium permanganate solution is added.	Potassium permanganate is not decolourised	Presence of unsaturated compound
6.	**Test For Amino Acid:** 2 drops of the extract is placed on a filter paper and dried well. 20ml of Biurette reagent is added.	violet colour developed	Absence of amino acids
7.	**Test For Type Of Compound**: 2ml of the extract is treated with 2 ml of dil.ferric chloride solution.	No colour change	Presence of Type of Compound

Preliminary Qualitative phytochemical tests procedure and interpretation of results

S.NO	PROCEDURE	INFERENCE
1.	**Calcium**	Presence of calcium
2.	**Sulphate**	presence of Sulphate
3.	**Chloride**	absence of Chloride
4.	**Carbonate**	presence of carbonate
5.	**Starch**	absence of starch
6.	**Iron**	Presence of iron
7.	**Phosphate**	Presence of phosphate
8.	**Tannic acid**	Presence of Tannic acid
9.	**Aluminium**	Presence of Aluminium
10.	**Magnesium**	absence of Magnesium
11.	**Ammonium**	presence of Ammonium
12.	**Mercury**	Absence of Mercury
13.	**Alkaloids**	Presence of Alkaloids
14.	**Reducing Sugar**	Presence of reducing sugar
15.	**Silicate**	Presence of Silicate
16.	**Copper**	Presence of Copper
17.	**Sodium**	Absence of Sodium
18.	**Lead**	Absence of Lead
19.	**Fluoride And Oxalate**	Absence of Fluoride and Oxalate

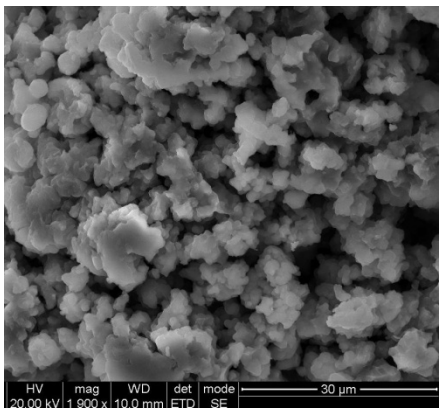
QUANTITATIVE ANALYSIS REPORT

SOPHISTICATED ANALYTICAL INSTRUMENT FACILITY

IITM, CHENNAI-36

PERKIN ELMER OPTIMA 5300DV ICP-OES

Sample ID- Atthippattaiyathi Kasayam



Analyte	Mean
As193.696	BDL
Al 308.215	BDL
Ca 317.933	246.249 mg/L
Cd 226.502	BDL
Cu 324.754	8.255 mg/L
Fe 238.204	16.985 mg/L
Hg253.652	BDL
K 766.491	120.159 mg/L
Mg 257.610	19.578 mg/L
Na 588.995	376.412 mg/L
P 214.914	77.995 mg/L
Pb 230.204	BDL
S 181.975	64.654 mg/L
Si 251.611	17.249 mg/L
Zn 213.856	23.756 mg/L

(BDL= below detection limit)

TOXICOLOGICAL EVALUVATION

ACUTE TOXICITY STUDY OF ATTHIPPATTAIYATHI KASAYAM

[WHO GUIDELINES, 1993]

Principle:

Acute toxicity was carried out in Swiss albino mice with a single exposure of 10 times of the recommended therapeutic dose of test compound the study duration will be 14 days.

Animal species : Swiss albino mice

Age / Weight / Size : 6 weeks. Mice-20-25 gms.

Gender : Both male and female

Number of Animals : Mice: 20

Acclimatization Period : 7 Days

Clinical dose : 15000 mg\day

S.No	Group	No of mice
1	Vehicle control	10 (5 male, 5 female)
2	Toxic dose (10 X therapeutic dose) (Single dose) (270 mg)	10 (5 male, 5 female)

Test Animals

Test animals were obtained from the animal laboratory of the King institute, Chennai and stocked at aninal house, National institute of siddha Chennai. All the animals were kept under standard environmental condition (27+ or – 2 degree c). The animals had free access to water and standard pellet diet (Sai meera foods pvt.ltd, Bangalore).The principles of laboratory animal care were followed and the Institutional Ethical Committee approved the use of animals and the study design. **(1248/ac/09/CPCSEA/4-07/ 2011)**

Route of administration: oral route was selected, because it is the normal route of clinical administration.

Test substance and vehicle

The Atthippattaiyathi kasayam powder is brown in colour with mild astringent taste. The test substance is soluble in water, in order to obtain and ensure the uniformity in drug distribution, the drug is extracted by decoction preparation method.

Administration of doses

Atthippattaiyathi kasayam decoction was administered to the group's in a single oral dose. The control groups were received equal volume of the vehicle. The animals were weighed before giving the drug. The dose level was calculated according to body weight, and surface area. Since the clinical dose was 15000mg\day It was converted to animal dose (270mg) and then administered. The principle of laboratory animal care was followed.

Observations

Observations were made and recorded systematically and continuously observed as per the guideline after substance administration. Animals were observed individually visual observations included skin changes, alertness, grooming, aggressiveness, sensitivity to sound, touch and pain, restlessness, tremors, convulsion, righting reflex, gripping reflex, pinna reflex, corneal reflex, writhing reflex, papillary reflex, urination, salivation, lacrimation for first 4 hrs, then periodically during the first 24 hrs. Animals were observed for body weight and mortality for 14 days. If animals dying during the period of study the animals were sacrificed. At the end of the 14^{th} day all animals were sacrificed and necropsy was done.

Body Weight

Individual weight of animals was determined before the test substance was administered and daily for 14 days. Weight changes were calculated and recorded. At the end of the test surviving animals were weighed and sacrificed.

Results:

Atthippattaiyathi kasayam at the dose 270mg/kg/bw did not exhibit any mortality in mice. No behavior changes were noted for the first 4 hours and for the next 24 hours and throughout the study period of 14 days. No weight reduction was noted before and after the acute study duration. Reflexes were found to be normal before and after the study. All other observations were found to be normal before and after the study. In Necropsy, the organs of the animal such as, Liver, Heart, Lungs, Pancreas, Spleen, Stomach, Intestine, Kidney, Urinary bladder, Uterus all appeared normal.

SUBACUTE TOXICITY STUDY OF ATTHIPPATTAIYATHI KASAYAM

[WHO GUIDELINES, 1993]

Animals : Male and Female wistar albino rats

Age : 6-8 weeks

Weight : 150-200 gms

Gender : Both male and female

Number of animals : Rat: 40

Acclimatization period : 7 Days

Clinical dose : 15000mg\day

Clinical duration : 28 days

S.No	Group	No of Rats
1	Vehicle control	10 (5male,5 female)
2	1XTherapeutic dose (270mg)	10 (5male,5 female)
3	5XTherapeutic dose (1350mg)	10 (5male,5 female)
4	10XTherapeutic dose(2700mg)	10(5male, 5 female)

Animal source:

Test animals were obtained from the animal laboratory of the King Institute, Chennai, and stocked at animal house National Institute of Siddha, Chennai. All the animals were kept under standard environmental condition (27+ or – 2 degree c). The animals had free access to water and standard pellet diet(Sai meera foods pvt.ltd, Bangalore). The principles of laboratory animal care were followed and the Institutional Ethical Committee approved the use of animals and the study design. **(1248/ac/09/CPCSEA/4-07/ 2011)**

Identification of animal: By cage number, animal number and individual marking on fur.

Housing & Environment: The animals were housed in polypropylene cages provided with bedding of husk. Dark and light cycle each of 12 hours.

Administration period:

The period of administration of the test substance to animals are depending on the expected period of clinical use. Since the clinical dose of the test drug is 28 days and as per WHO guidelines the administration period is reported to be 6 months.

Dose selection:

The results of acute toxicity studies in swiss albino mice indicated that Atthippattaiyathi kasayam was non toxic and no behavioral changes, mortality was observed. On the basis of these results, the doses were selected for the study as per WHO guidelines.

Preparation and administration of dose:

Atthippattaiyathi kasayam was extracted by decoction preparation method. It was administered to animals at dose levels of 1X therapeutic dose (270mg), 5X Therapeutic dose (1350mg) and 10X Therapeutic dose (2700mg). The control animals were administered vehicle only. Administration was by Oral Gavage once a day for 28 days.

METHODOLODY:

Randomization, numbering and grouping of animal:

The animals were randomly divided into four groups for dosing up to 28 days. Each group consist of 10 animals (5 per sex in each group) were allowed acclimatization period of 7 days to laboratory conditions prior to the initiation of treatment. Each animal was fur marked with picric acid. The females were nulliparous and non pregnant.

OBSERVATION:

Experimental animals were kept under observation throughout the course of study for the following,

Body weight:

Weight of each rat was recorded on day 1 and at weekly intervals throughout the course of study and at termination to calculate relative organ weights. From the data mean body weights and percent body gain were calculated.

Food and water consumption:

The quantity of food consumed by groups consisting of an animal for different doses was recorded at weekly intervals. Food consumed per animal was calculated for control and the treated dose groups

Clinical sings

All animals were observed daily for clinical sings. The time of onset intensity and duration of this symptom if any were recorded

Mortality:

All animals were observed twice daily for mortality during entire course of study.

TERMINAL STUDIES:

NECROPSY:

All the animals were sacrificed on day 91 under ether anesthesia. Necropsy of all animals was carried out and the weights of the organs including liver, kidneys, brain, heart, and lungs were recorded.

HISTOPATHOLOGY:

Tissue samples of organs from control and treated animals were preserved in 10% formalin for preparation of sections using microtome. The organs included liver, kidneys, heart, lungs and stomach of the animals were preserved and they were subjected to histopathological examination.

The organ pieces (3-5 micron) were fixed in 10% formalin for 24 hours and washed in running water for 24 hours. Samples were dehydrated in tissue processor and then cleaned in benzene to remove absolute alcohol. Embedding was done by passing the cleared sample through three cups containing molten paraffin at 50 degree Celsius and then a cubical block of paraffin made by the L moulds it was followed by microtome and the slides were stained with haematoxylin–eosin stain. Stained sections of each organ were examined under light microscope at high (40X) power magnification. All the histopathological slides were prepared at Dept of Pathology, Vels University, pallavaram, Chennai.

Results:

<u>270MG TREATED (Low dose)</u>

Kidney: shows normal renal tissue with glomeruli and tubules.

Spleen: shows normal spleen with lymphoid aggregation.

Liver: shows almost normal hepatocytes and occasional binucleate cells.

Stomach: shows normal mucosal glands.

Lung: shows normal alveoli.

Heart: shows normal cardiac muscle bundles.

Pancrea: shows normal acini with islets of β-cells

1350MG TREATED (Mid dose)

Kidney: shows renal tissue with focal tubular damage, interstitial inflammatory collection. Glomeruli shows epithelial proliferation.

Liver: shows hepatocytes with focal mild fatty change.

Spleen: shows congestion with lymphoid hyperplasia.

Stomach: shows near normal mucosal gland with mild exudates.

Lung: shows congested alveolar wall with mild thickening and mild emphysematous changes.

Pancreas: shows pancreas with acini and normal islets.

Heart: shows congestion and mild inflammatory infiltration in between cardiac muscle bundles.

2700MG TREATED (High dose)

Stomach: shows stomach with superficial erosion and congestion.

Heart: shows hypertrophic cardiac muscle bundles.

Spleen: shows lymphoid hyperplasia.

Liver: shows marked dilatation of sinusoids, degeneration of hepatocytes, necrosis.

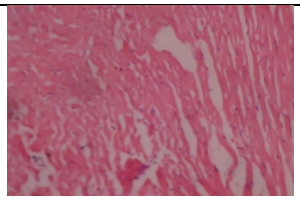
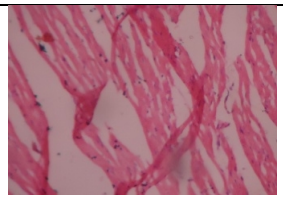
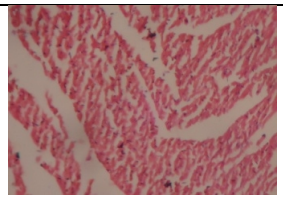
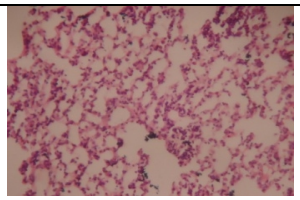
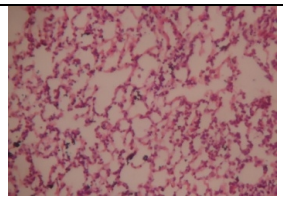
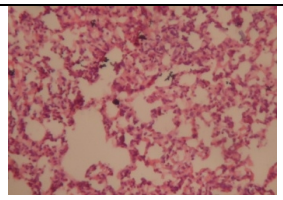
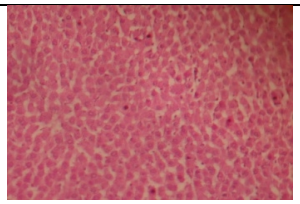
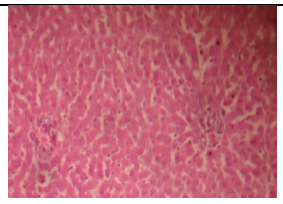
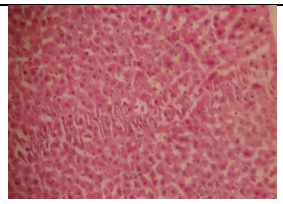
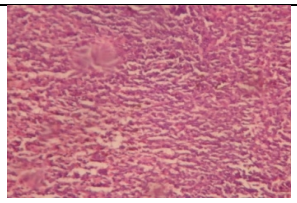
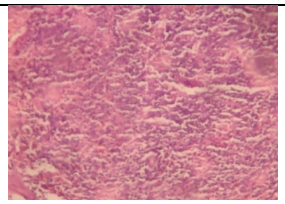
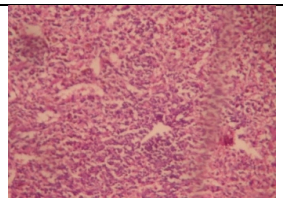
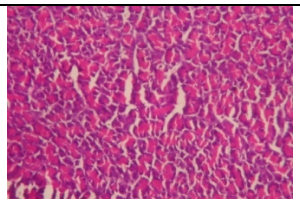
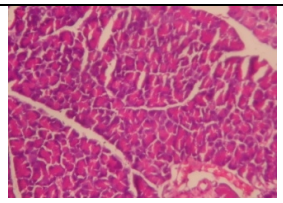
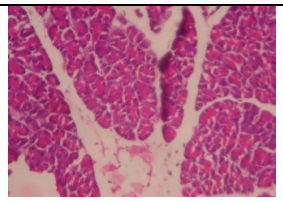
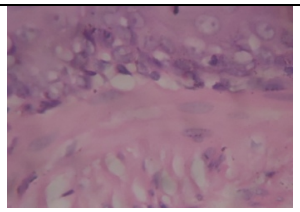
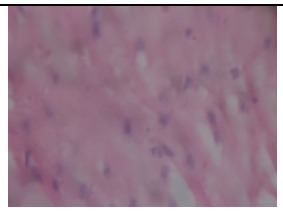
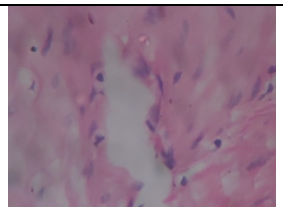
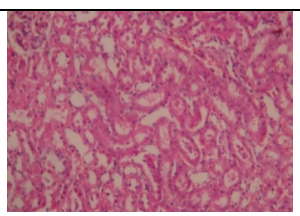
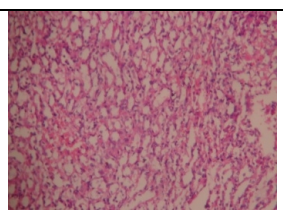
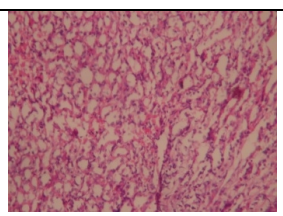
Kidney: shows renal tissue with tubular epithelial damage.

Pancreas: shows atrophic islet cells.

Lung: shows congestion, narrowed alveolar space and thickened alveolar wall.

Impression: Normal study

HISTOPATHOLOGY SLIDES

Slides	Control Group	5x Group	10x Group
Heart			
Lung			
Liver			
Spleen			
Pancreas			
Stomach			
Kidney			

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASA PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "MATHUMEGAM" (TYPE-2 DIABETES MELLITUS) AND THE DRUG OF CHOICE IS "ATTHIPPATTAIYATHI KASAYAM" (INTERNAL)

FORM-l (SCREENING & SELECTION PROFORMA)

IEC NO: NIS/IEC/2011/3/07

1. SI NO: _____ 2.OP/IP NO: _____
3.NAME:_____

4.RELIGION: H/C/M/O 5. AGE: _____ 6.GENDER: _____

7. OCCUPATION:_____ 8. INCOME: _____ 9. CONTACT NO: _____

10. ADDRESS:
_____

INCLUSION CRITERIA	EXCLUSION CRITERIA
• Age : 30-55Yrs – Yes/No • Sex: Male/Female **Symptoms:** • Polyuria – Yes/No • Nocturia – Yes/No • Polydipsia – Yes/No • Polyphagia – Yes/No • Body pain – Yes/No • Weight gain (obesity) – Yes/No • Tiredness – Yes/No • Burning feet – Yes/No • Genital pruritus – Yes/No **Blood glucose level:** • Fasting plasma glucose level- 126 to180 mg/dl – Yes/No • 2 hours plasma glucose level- 200 to 300 mg/dl – Yes/No	• IDDM (Insulin Dependent Diabetes Mellitus) – Yes\No • Diabetic complications like micro and macrovascular complications etc -Yes\No • Cardiac diseases – Yes\No • Pulmonary diseases – Yes\No • Renal diseases – Yes\No • Thyroid dysfunctions – Yes\No • Gestational diabetes – Yes\No • Other endocrine abnormalities – Yes\No • Patient who are not willing to give blood sample – Yes\No

Asymptomatic individuals fulfilling the following criteria may be screened

- Previously identified Impaired Fasting Glucose (IFG) or Impaired Glucose Tolerance (IGT).

  IFG- FPG >110 and <126mg/dl – Yes/No

  IGT- 2h PG >140 and <200mg/dl – Yes/No
- Family history of diabetes – Yes/No
- Sedentary lifestyle – Yes/No
- History of gestational diabetes mellitus, recurrent fetal loss or delivery of large baby ≥ 3.5kg – Yes/No
- Dyslipidemia – Yes/No
- Hypertension (>140/90 mm Hg in adults) – Yes/No
- Urine test – glycosuria, microalbuminuria – Yes/No
- Acanthosis nigricans – Yes/No
- Patient willing to sign the informed consent stating that he will conscientiously stick to the treatment during 40 days but can opt out of the trial of his own conscious discretion – Yes/No
- Patients who are willing to provide blood and urine for lab investigation – Yes/No

Admited to trail 1.Yes ☐ 2.No ☐

If Yes, Serial NO: _____

DATE: **STATION:**

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF THE LECTURER

SIGNATURE OF THE HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASA PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "MATHUMEGAM" (TYPE-2 DIABETES MELLITUS) AND THE DRUG OF CHOICE IS "ATTHIPPATTAIYATHI KASAYAM" (INTERNAL)

FORM I-A HISTORY PROFORMA

IEC NO: NIS/IEC/2011/3/07

1. Serial No of the patient: _____ OP.NO/IP.NO:_____

2. Name: _____ 3. Gender: F/M

4. Age (years): _____ DOB ☐☐ ☐☐ ☐☐☐☐
Date Month Year

5. Address: _____

6. Occupation: _____

7. Educational Status: A) Illiterate ☐ B) Literate ☐

8. Height:_____cms 9.Weight:_____kg 10. BMI: _____ kg/m²

11. Marital status: 1.Married ☐ 2.Unmarried ☐

10. **Complaints and Duration:**

_____
_____
_____
_____

11. **History of present illness:**

_____
_____
_____
_____

12. **Past History:**

_____
_____
_____
_____

13. **Socio economic status:**
Income group 1.lower ☐ 2.middle ☐ 3.higher ☐

14. **Treatment History:**

Had the patient been treated before with Allopathy drug? Yes ☐ No ☐

	1.Yes	2.No
Insulin injection	☐	☐
Oral antidiabetic drugs	☐	☐

How long: _____

15. Family history:

Whether diabetes mellitus runs this family?

1) Yes ☐ 2) No ☐

If yes, mention the relationship of affected person(s)

1. _____
2. _____
3. _____

16. Habit of

A) Smoking	1. Yes; duration _____ years; Quantity _____ ___	2.No
B) Tobacco chewing	1. Yes; duration _____ years	2.No
C) Betel chewing	1. Yes; duration _____ years	2.No
D) Alcoholism	1. Yes; duration _____ years; Quantity_____ml	2.No

17.Dietry style: A.) Pure vegetarian B.) Non-vegetarian C.) Mixed diet

DATE: **STATION:**

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF THE LECTURER

SIGNATURE OF THE HOD

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PRE CLINICAL AND CLINICAL STUDY ON "MATHUMEGAM" (TYPE-2 DIABETES MELLITUS) AND THE DRUG OF CHOICE IS "ATTHIPPATTAIYATHI KASAYAM" (INTERNAL)
FORM-II AND II-A CLINICAL ASSESSMENT ON ENROLLMENT AND ON VISITS
IEC NO: NIS/IEC/2011/3/07

1. S NO: _____ 2. OP/IP NO: _____

3. NAME: _____ 4.GENDER: M/F

5. DATE OF ASSESSMENT : _____

Initial (0th day) ☐ **10th day** ☐ **20th day** ☐ **30th day** ☐ **40th day** ☐

SIDDHA SYSTEM OF EXAMINATION

1. ENVAGAI THERVU: [EIGHT-FOLD EXAMINATION]

I. NAADI: [PULSE PERCEPTION]

	0th day	10th day	20th day	30th day	40th day		0th day	10th day	20th day	30th day	40th day
Vali						Iyya vali					
Azhal						Vali Iyyam					
Iyyam						Azhal Iyyam					
Vali Azhal						Iyya Azhal					
Azhal vali											

II. NAA:[TONGUE]

	0th Day	**10th Day**	**20th Day**	**30th Day**	**40th Day**
Colour	Dark / Yellow/Red / Pale/Normal	Dark/Yellow/ Red/Pale/Normal	Dark/Yellow/ Red/Pale/Normal	Dark/Yellow/ Red/Pale/Normal	Dark/Yellow/ Red/Pale/Normal
Taste	Sweet/Bitter/Sour Pungent/None	Sweet/Bitter/Sour Pungent/None	Sweet/Bitter/Sour Pungent/None	Sweet/Bitter/Sour Pungent/None	Sweet/Bitter/Sour Pungent/None
Coating	Present/Absent	Present/Absent	Present/Absent	Present/Absent	Present/Absent
Fissure	Present/Absent	Present/Absent	Present/Absent	Present/Absent	Present/Absent
Saliva	Normal/Increased/ Decreased	Normal/Increased Decreased	Normal/Increased/ Decreased	Normal/Increased/ Decreased	Normal/Increased/ Decreased

Dryness	Present/Absent	Present/Absent	Present/Absent	Present/Absent	Present/Absent
Glossitis	Present/Absent	Present/Absent	Present/Absent	Present/Absent	Present/Absent
Baldness	Present/Absent	Present/Absent	Present/Absent	Present/Absent	Present/Absent

III.NIRAM: [COMPLEXION]

0^{th} Day	10th day	20th day	30th day	40^{th} Day
Dark/Yellow tinted/Whitish brown / Pale	Dark/Yellow tinted / Whitish brown / Pale	Dark/Yellow tinted / Whitish brown/ Pale	Dark/Yellow tinted / Whitish brown/ Pale	Dark/Yellow tinted/ Whitish brown / Pale

IV.MOZHI: [VOICE]

0^{th} Day	10th day	20^{th} Day	30^{th} Day	40^{th} Day
Medium/High Low pitched	Medium/High/ Low pitched	Medium/High/ Low pitched	Medium/High/ Low pitched	Medium/High/ Low pitched

V.VIZHI: [EYES] (Lower palpabrel conjunctiva)

0^{th} Day	10th day	20^{th} Day	30^{th} Day	40^{th} Day
Yellow Red/ Pale/Normal	Yellow Red/ Pale/Normal	Yellow Red/ Pale/Normal	Yellow Red/ Pale/Normal	Yellow Red/ Pale/Normal

VI. MALAM; [BOWEL HABITS / STOOLS]

Malam	0^{th} Day	10^{th} Day	20^{th} Day	30^{th} Day	40^{th} Day
Colour	Dark/ Yellow/ Pale/Others	Dark/ Yellow/ Pale	Dark/ Yellow Pale	Dark/ Yellow Pale	Dark/ Yellow Pale
Consistency	Solid/Semisolid Watery	Solid/Semisolid Watery	Solid/Semisolid Watery	Solid/Semisolid Watery	Solid/Semisolid Watery
Stool bulk	Normal/Reduced	Normal/Reduced	Normal/Reduced	Normal/Reduced	Normal/Reduced
Constipation	Present/Absent	Present/Absent	Present/Absent	Present/Absent	Present/Absent
Diarrhoea	Present/Absent	Present/Absent	Present/Absent	Present/Absent	Present/Absent

VII. URINE EXAMINATION:

NEERKURI	0th Day	10th Day	20th Day	30th Day	40th Day
Niram [Colour]	White/Yellowish/ Straw coloured/ Crystal clear	White/Yellowish/ Straw coloured Crystal clear	White/Yellowish/ Straw coloured/ Crystal clear	White/Yellowish/ Straw coloured/ Crystal clear	White/Yellowish/ Straw coloured/ Crystal clear
Manam [Odour]	Present Absent	Present Absent	Present Absent	Present Absent	Present Absent
Nurai [Froth]	Nil Reduced/Increased	Nil Reduced/Increased	Nil Reduced/Increased	Nil Reduced/Increased	Nil Reduced/Increased
Edai [Sp.gra]	Normal Increased/Reduced	Normal Increased/Reduced	Normal Increased/Reduced	Normal Increased/Reduced	Normal Increased/Reduced
Enjal [Deposits]	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Volume	Normal Increased/Reduced	Normal Increased/Reduced	Normal Increased/Reduced	Normal Increased/Reduced	Normal Increased/Reduced

NEIKURI	0th day	10th day	20th day	30th day	40th day
Serpentine fashion					
Annular/Ringed fashion					
Pearl beaded fashion					
Mixed fashion					
Other fashion					

VIII. SPARISAM: [PALPATORY PERCEPTION]

0th Day	10th Day	20th Day	30th Day	40th Day
Warmth/Cold/Normal Sweat	Warmth/ Cold/Normal Sweat	Warmth/ Cold/Normal Sweat	Warmth/ Cold/Normal Sweat	Warmth/ Cold/Normal Sweat

5. THEGI: [TYPE OF BODY CONSTITUTION]

Vatham predominant		Kabam predominant	
Pitham predominant		Thondha udal	

6.NILAM: [LAND WHERE PATIENT LIVED MOST]

Kurinji	Mullai	Marutham	Neithal	Palai
(Hilly terrain)	(Forest range)	(Plains)	(Coastal belt)	(Arid regions)

7. KAALAM

Kaarkalam-		Pinpanikalam	
Koothirkalam-		Ilavenil	
Munpanikalam -		Muthuvenil	

8. GUNAM

Sathuvam	Rasatham	Thamasam

9. AIMPORIGAL (SENSORY ORGANS)

AIMPORIGAL	0^{th} day	10^{th} day	20^{th} day	30^{th} day	40^{th} day
Mei (Skin)					
Vai (Buccal Cavity)					
Kann (Eye)					
Mooku (Nose)					
Sevi (Ear)					

10.KANMENDRIYAM (MOTOR ORGANS)

	0^{th} day	10^{th} day	20^{th} day	30^{th} day	40^{th} day
Kai (upper limb)					
Kaal (lower limbs)					
Vai (buccal cavity)					
Eruvai (excretory organs)					
Karuvai (reproductive organs)					

11. KOSANGAL (Sheath)

	0^{th} day	10^{th} day	20^{th} day	30^{th} day	40^{th} day
Annamaya Kosam					
Pranamaya kosam					
Manomaya kosam					
Vignanamaya kosam					
Ananthamaya kosam					

12. MUKKUTRAM: [AFFECTION OF THREE HUMORS]

A)VATHAM:

	0^{th} day	10^{th} day	20^{th} day	30^{th} day	40^{th} day
Praanan					
Abaanan					
Samaanan					
Udhaanan					
Viyaanan					
Naagan					
Koorman					
Kirukaran					
Devathathan					
Dhananjeyan					

B) PITHAM:

	0th day	10th day	20th day	30th day	40th day
Anarpitham					
Prasakam					
Ranjakam					
Aalosakam					
Saathakam					

C) KABAM:

	0th day	10th day	20th day	30th day	40th day
Avalambagam					
Kilethagam					
Pothagam					
Tharpagam					
Santhigam					

13. SEVEN DHATHUS: (7 SOMATIC COMPONENTS)

	0th day	10th day	20th day	30th day	40th day
Saaram [Chyme]					
Senneer [Blood]					
Oon [Muscle]					
Kozhuppu [Fat]					
Enbu [Bones]					
Moolai [Bonemarrow]					
Sukkilam/Suronitham [Genital discharges]					

14. SYSTEMIC EXAMINATION:

	0^{th} day	10^{th} day	20^{th} day	30^{th} day	40^{th} day
LOCOMOTOR SYSTEM					
CARDIO VASCULAR SYSTEM					
RESPIRATORY SYSTEM					
GASTRO INTESTINAL SYSTEM					
CENTRAL NERVOUS SYSTEM					
UROGENITAL SYSTEM					
ENDOCRINE SYSTEM					

15. GENERAL EXAMINATION:

	0^{th} day	10^{th} day	20^{th} day	30^{th} day	40^{th} day
Height (cms)					
Weight (kg)					
Temperature(°F)					
Pulse rate (per min)					
Heart rate (per min)					
Respiratoryrate(per min)					
Blood pressure(mm/Hg)					
Pallor					
Jaundice					

Cyanosis					
Lymphadenopathy					
Pedal edema					
Clubbing					
Jugular vein pulsation					

16. CLINICAL SYMPTOMS

S.NO	CLINICAL SYMPTOMS	0^{th} day	10^{th} day	20^{th} day	30^{th} day	40^{th} day
1.	POLYURIA					
2.	NOCTURIA					
3.	POLYDIPSIA					
4.	POLYPHAGIA					
5.	BODY PAIN					
6.	WEIGHT GAIN (obesity)					
7.	TIREDNESS					
8.	BURNING FEET					
9.	GENITAL PRURITUS					

DATE:

STATION:

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF THE LECTURER

SIGNATURE OF THE HOD

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASA PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "MATHUMEGAM" (TYPE-2 DIABETES MELLITUS) AND THE DRUG OF CHOICE IS "ATTHIPPATTAIYATHI KASAYAM" (INTERNAL)

FORM-III LABORATORY PARAMETERS-CHART

IEC NO: NIS/IEC/2011/3/07

1. OP/IP No: _____ 2.S. No: _____

3. Name: _____ 4. Age: _____ years 5.Gender: M/F

BLOOD INVESTIGATION		NORMAL VALUES	BEFORE TMT Date:	AFTER TMT Date:
HB(gms%)		M:13-18 W:11-16		
T.RBC(milli/cu.mm)		M:4.5-6.5 W:3.5-5.5		
ESR (mm)	½ hr.	-		
	1 hr.	M:0-10 W:0-20		
T.WBC (/cu.mm)		4000-11000		
DIFFERENTIAL COUNT (%)	Polymorphs	40-75		
	Lymphocytes	20-35		
	Monocytes	2-10		
	Esonophils	1-6		
	Basophils	0-1		

Blood Investigation		Normal Values	Before TMT Date:	After TMT Date:
Lipid profile (mg/dl)	Serum cholesterol	150-250		
	HDL	30-60		
	LDL	Upto 130		
	VLDL	40		
	TGL	Upto 160		

RFT (mg/dl)	**Blood urea**	**16-50**		
	Serum creatinine	**0.6-1.2**		
	Serum Uric acid	**M:3-9 W: 2.5-7.5**		
LFT (mg/dl)	**Total bilirubin**	**0.3-1**		
	Direct bilirubin	**0.1-0.3**		
	Indirect bilirubin	**0.2-0.8**		
	Serum total protein	**6-8**		
	Serum Albumin	**3.5-5.5**		
	Serum globulin	**2-3.5**		
	Serum calcium	**9-11**		
	Serum phosphorous	**2-5**		
	SGOT (IU/L)	**6-18**		
	SGPT (IU/L)	**3-26**		
	Alkaline phosphatase (IU/L)	**80-290**		

URINE INVESTIGATION	**Before TMT(with Date)**	**After TMT (With Date)**
Albumin		
Fasting sugar		
PP sugar		
Deposits		

SPECIFIC INVESTIGATION:

OGTT

BLOOD GLUCOSE TEST	NARMAL VALUE	BEFORE TMT	AFTER TMT
OGTT **FASTING (OVER NIGHT 8-10hr)**	**<126 mg/dl**		
2 HOURS AFTER GLUCOSE LOAD	**<200 mg/dl**		

DATE: **STATION:**

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF THE LECTURER

SIGNATURE OF THE HOD

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PRE CLINICAL AND CLINICAL STUDY ON "MATHUMEGAM" (TYPE-2 DIABETES MELLITUS) AND THE DRUG OF CHOICE IS "ATTHIPPATTAIYATHI KASAYAM" (INTERNAL)
FORM IV –C (DRUG COMPLIANCE FORM)
IEC NO: NIS/IEC/2011/3/07

Name:_____ OP/IP No: Serial No: DRUG NAME: _____

On 0th day-Date: Drugs issued: **DOSE:_____** (before food)
On 10th day-Date: Drugs issued: Drugs returned:
On 20th day-Date: Drugs issued: Drugs returned:
On 30th day-Date: Drugs issued: Drugs returned:
On 40th day-Date: Drugs returned:

Day	Date	Morning (7-8 am)	After noon (1-2 pm)	Evening (7-8 pm)
Day 1				
Day2				
Day3				
Day4				
Day5				
Day6				
Day7				
Day8				
Day9				
Day10				
Day11				
Day12				
Day13				
Day14				
Day15				
Day16				
Day17				
Day18				
Day19				
Day20				
Day 21				
Day22				
Day23				
Day24				
Day25				

Day26				
Day27				
Day28				
Day29				
Day30				
Day31				
Day32				
Day33				
Day34				
Day35				
Day36				
Day37				
Day38				
Day39				
Day40				

DATE:

STATION:

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF THE LECTURER

SIGNATURE OF THE HOD

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PRE CLINICAL AND CLINICAL STUDY ON "MATHUMEGAM" (TYPE-2 DIABETES MELLITUS) AND THE DRUG OF CHOICE IS "ATTHIPPATTAIYATHI KASAYAM" (INTERNAL)

FORM IV-D DIETARY ADVICE FORM
IEC NO: NIS/IEC/2011/3/07

DIET ADVICE:

Don't

- Avoid sugar, honey, jaggery, sweets, very sweet fruits, fruit juices, roots, tubers, ghee, milk, curd, oily foods, chicken, mutton, egg yolk.
- Avoid alcohol, smoking, tobacco, betel nut.

Do's

- Main source should be cereals, mixed coarse grains, whole pulses, salads and soybeans.
- Take Protein from vegetables sources like tender fresh vegetables, fiber content vegetables, greens and butter milk, sea fish, lean meat.
- Fiber rich food include whole grains, whole pulses, soybean, green leafy vegetables and fenu-greek seeds.
- Ground nut, sesame, cotton seed, rice bran and safflower oils should be used.
- Salt up to 6g/day is permitted. Restrict pickles, papad, chatni and salty processed foods,
- Brisk walking for 45 minutes daily.

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை-47
அயோத்திதாசர் பண்டிதர் மருத்துவமனை
பொதுமருத்துவத் துறை
மதுமேகம் (வகை-2 டயபிடிஸ் மெல்லிடஸ்) நோய்க்கான சித்த மருந்தின்
(அத்திப்பட்டையாதி கசாயம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான
படிவம் IV-D உணவு பரிந்துரை படிவம்
IEC NO: NIS/IEC/2011/3/07

தவிக்க வேண்டியவை:

- சர்கரை, தேன், பனைவெல்லம், இனிப்பு வகைகள், மிகு இனிப்பான பழங்கள், பழச்சாறு வகைகள், கிழங்கு வகைகள், நெய், பால், தயிர், எண்ணெய் பலகாரங்கள், கோழி, ஆடு, முட்டை மஞ்சள்கரு ஆகியவற்றை தவிர்கவும்.
- மது, புகை, வெற்றிலை, பாக்கு இவற்றை தவிர்கவும்.

சேர்க்க வேண்டியவை:

- தானிய கலவை, முழைகட்டிய தனியம், பருப்பு வகைகள், பச்சடி வகை, சோயாபீன் ஆகியவை முக்கிய உணவுகள் ஆகும்.
- பச்சை இளம்காய்கள், நார்சத்துக் காய்கள், கீரைகள் மற்றும் மோர், கடல்மீன், கொழுப்பற்ற மாமிசம் ஆகியவற்றிலிருந்து புரதங்களை பெறுவது நன்று.
- முழுதானியங்கள், முழுபருப்பு வகைகள், சோயாபீன், பச்சை இலைக்காய்கறிகள் மற்றும் வெந்தயம் ஆகியவை நார்சத்துமிக்க உணவுகள்.
- கடலை எண்ணெய், நல்லெண்ணெய், பருத்திவிதை எண்ணெய், ரைஸ்பிரன் எண்ணெய், சாபிளார் எண்ணெய் ஆகிய எண்ணெய் வகைகளை பயன்படுத்தவும்.
- தினசரி 6கி உப்பு மட்டுமே எடுத்துக்கொள்ளவும், ஊறுகாய், அப்பளம், சட்டினி மற்றும் உப்பில் ஊறிய தயாரிப்பு உணவுகளை தவிர்கவும்.
- தினசரி 45 நிமிடம் வேக நடை நடப்பது நன்று.

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FORM IV-B WITHDRAWAL FORM

IEC NO: NIS/IEC/2011/3/07

NAME: _____ **OPD/ IPD NUMBER:** _____

AGE: _____ **SERIAL NO:** _____

DATE OF TRIAL COMMENCEMENT:

DATE OF WITHDRAWAL FROM TRIAL:

REASONS FOR WITHDRAWAL:

- Long absence at reporting : Yes/ No
- Irregular treatment: Yes/ No
- Shift of locality : Yes/No
- Increase in severity of symptoms: Yes/No
- Development of severe adverse drug reactions: Yes/No

DATE:

STATION:

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF THE LECTURER

SIGNATURE OF THE HOD

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FORM IV-E ADVERSE DRUG REACTION FORM

IEC NO: NIS/IEC/2011/3/07

NAME: _____ OPD/ IPD NUMBER: _____

AGE: _____ SERIAL NO: _____

DATE OF TRIAL COMMENCEMENT:

DATE OF WITHDRAWAL FROM TRIAL:

DESCRIPTION OF ADVERSE REACTION:

_____

_____

_____

_____

DATE:

STATION:

SIGNATURE OF THE INVESTIGATOR

SIGNATURE OF THE LECTURER

SIGNATURE OF THE HOD

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CONSENT FORM-IV A

IEC NO: NIS/IEC/2011/3/07

CERTIFICATE OF CONSENT

"I have read the foregoing information, or it has been read to me. I have had the opportunity to ask question about it and any questions I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care."

"I have received a copy of the information sheet/consent form."

Date: Signature of the participant

In case of illiterate participant

"I have witnessed the reading of the consent form to the potential participant, and the individual has had the opportunity to ask question. I confirm that the individual has given consent freely."

Date:

Signature of a witness

Left thumb impression of participant
(Selected by the participant bearing no connection with the project team)

Date: Signature of the Doctor

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை-47
அயோத்திதாச பண்டிதர் மருத்துவமனை
பொதுமருத்துவத் துறை
மதுமேகம் (வகை-2 டயபிடிஸ் மெல்லிடஸ்) நோய்க்கான சித்த மருந்தின் (அத்திப்பட்டையாதி கசாயம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான
ஒப்புதல் படிவம்-IV A
IEC NO: NIS/IEC/2011/3/07
ஒப்புதல்படிவ சான்றிதல்

"நான் மேற்கூறிய தகவல் படிவத்தை படித்து அல்லது படிக்க கேட்டு கொண்டேன். இது தொடர்பான விளக்கங்களையும் கேட்டு தெரிந்து கொண்டேன். எந்தவித வற்புறுத்தலின்றி என் சொந்த விருப்பத்தின் பேரில் என்னை இந்த ஆராய்ச்சிக்கு உட்படுத்த என் முழுமனதோடும் சுயநினைவோடும் சம்மதம் தெரிவிக்கிறேன். நான் இந்த மருத்துவ ஆய்வின் போது காரணம் எதுவும் கூறாமல் எப்பொழுது வேண்டுமானாலும் இந்த ஆய்விலிருந்து என்னை விடுவித்துக்கொள்ளும் உரிமையை தெரிந்திருக்கின்றேன்."

நான் தகவல் படிவம்/ஒப்புதல் படிவ நகலை பெற்றுக்கொண்டேன்.

தேதி: கையொப்பம்:

எழுதபடிக்க தெரியாத நோயாளியாக இருக்கும் பட்சத்தில்

"எனது முன்னிலையில் முதன்மை ஆய்வாளரால் இந்த ஆய்வு குறித்து அனைத்து தகவல்களும் நோயாளிக்கு தெளிவாக விளக்கப்பட்டது. ஆய்வு குறித்த நோயாளியின் சந்தேகங்களுக்கு கேள்வி எழுப்ப வாய்ப்பு வழங்கப்பட்டது. நோயாளி முழுமனதுடன் எந்தவித வற்புறுத்தலின்றி இந்த ஆய்வில் பங்கேற்க்கிறார் என்று சாட்சியளிக்கிறேன்".

தேதி:

சாட்சிக்காரர் கையொப்பம்:

நோயாளியின் இடதுகை பெருவிரல் ரேகை

தேதி: மருத்துவர் கையொப்பம்

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47
AYOTHIDASA PANDITHAR HOSPITAL
DEPARTMENT OF MARUTHUVAM
PRE CLINICAL AND CLINICAL STUDY ON "MATHUMEGAM" (TYPE-2 DIABETES MELLITUS) AND THE DRUG OF CHOICE IS "ATTHIPPATTAIYATHI KASAYAM" (INTERNAL)
FORM IV – INFORMATION SHEET
IEC NO: NIS/IEC/2011/3/07

Name of the Principal Investigator: Dr.S.G.Senthil kumar

Name of the Institution : National Institute of Siddha
Tambaram Sanatorium
Chennai-47.

- *I, Dr.S.G.Senthil kumar studying M.D(Siddha) in National Institute of Siddha, Chennai. The disease called* ***Mathumegam (Type-2 Diabetes Mellitus)*** *becomes heterogeneous group of metabolic disorders characterized by hyperglycemia resulting from defects in insulin secretion, insulin resistance or both. It includes the symptoms like, frequent urination, increased thirst, increased hunger, general tiredness and burning feet. This condition is being treated in NIS with many siddha formulations. As a part of M.D(Siddha) research programme and developing new efficacious medicine, we propose to study the* ***Atthippattaiyathi kasayam*** *formulation for treating the condition. This formulation has been mentioned in siddha literature and empirical evidence with contemporary tools is required for documentation. You can receive medicines free of cost. The duration of treatment period is 40 days. You have to visit NIS every 10 days and collect drugs for 10 days. The diagnosis tests will be carried out free of cost. We will assess the effect of treatment after completion of 40 days of treatment using clinical and lab parameters.*

- *In this regard, we need to ask you few questions. We will maintain confidentiality of your comments and data obtained from you. There will be no risk of disclosing your identity and no physical, psychological or professional risk is involved by taking part in this study.*
- *Taking part in this study is voluntary. No compensation will be paid to you for taking part in this study. You can choose not to answer any specific question. There is no specific benefit for you if you take part in the study, but you will be under our clinical monitoring and specific attention will be given for your health. Taking part in the study may be of benefit to the community, as it may help us to develop medicine for Mathumegam (Type-2 Diabetes Mellitus). You can withdraw from the study at the midst of treatment period, if you are not interested to continue and you will receive our usual treatment without condition.*
- *The information we will collect in this study, will remain between you and the principal investigator. We will ask you a few questions through questionnaire. We will not write your name on different forms which sent to different investigating/analysis sections and we will use a code instead given by the principal investigator. Only the principal investigator will know the key to this code which will be kept in safe custody. If you agree to be a participant in this study, you will be screened as per the study protocol.*

- *If you wish to find out more about this study before taking part, you can ask me all the questions you want or contact Dr.S.G.Senthil kumar, PG scholar cum principal investigator of this study, attached to the National Institute of Siddha, Chennai (Mobile phone no:9994172837). You can also contact the Chairman/Member-secretary of Ethics committee, National Institute of Siddha, Chennai – 600047, Tel no: 91-44-22411611, for rights and participation in the study.*

தேசிய சித்த மருத்துவ நிறுவனம், சென்னை 47
அயோத்திதாச பண்டிதர் மருத்துவமனை
பொதுமருத்துவத் துறை
மதுமேகம் (வகை-2 டயபிடிஸ் மெல்லிடஸ்) நோய்க்கான சித்த மருந்தின் (அத்திப்பட்டையாதி கசாயம்) பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கான தகவல் படிவம்.

FORM IV B - தகவல் படிவம்

IEC NO: NIS/IEC/2011/3/07

முதன்மை ஆராய்ச்சியாளர் பெயர் : **Dr. ச.கோ.செந்தில் குமார்**
நிறுவனத்தின் பெயர் : **தேசிய சித்த மருத்துவ நிறுவனம்**
தாம்பரம் சானட்டோரியம்
சென்னை- 47

Dr.ச.கோ.செந்தில் குமார் ஆகிய நான் தேசிய சித்த மருத்துவமனையில் பட்ட மேற்படிப்பு பயின்று வருகிறேன். **மதுமேகம் (வகை-2 டயபிடிஸ் மெல்லிடஸ்)** என்னும் வளர்சிதைமாற்ற நோயானது கணையம் என்னும் நாளமில்லா சுரப்பியின் இன்சுலின் என்னும் ஹார்மோன் சுரப்பின் பயன்பாட்டுத் தடையின் காரணமாகவோ, இன்சுலின் குறைபாட்டின் காரணமாகவோ அல்லது இரண்டின் காரணமாகவோ குருதியில் சர்க்கரையின் அளவை அதிகப்படுத்தும் ஒரு நோயாகும். இந்நோய் அடிக்கடி சிறுநீரிழிதல், அதிகபசி, அதிதாகம், உடல் சோர்வு, அசதி, கால் எரிச்சல் போன்ற குறிகுணங்களைத் தோற்றுவிக்கும். இந்நோய்க்கு தேசிய சித்த மருத்துவமனையில் பல சித்த மருந்துகள் பயன்படுத்தப்பட்டு வருகின்றன. சித்த மருத்துவ பட்டமேற்படிப்பில், ஆய்வின் ஒரு பகுதியாக புதிய மருந்துகளை பயன்படுத்தும் நோக்கில் **"அத்திப்பட்டையாதி கசாயம்"** என்னும் மருந்தினை இந்நோய்க்கு வழங்க பரிந்துரை செய்கிறோம். இந்த மருந்தின் செய்முறை, அளவு மற்றும் மருத்துவ பயன்கள் அனைத்தும் அங்கீகரிக்கப்பட்ட சித்த மருத்துவ நூலில் கூறப்பட்டுள்ளது. எந்தவித கட்டணமுமின்றி தாங்கள் இந்த மருந்தினை பெற்றுக்கொள்ளலாம். இந்த ஆய்வில் மருந்து உட்கொள்ளும் காலம் 40 நாட்கள் ஆகும். 10 நாட்களுக்கு ஒருமுறை தேசிய சித்த மருத்துவமனைக்கு நேரில் வந்து 10 நாட்களுக்கான மருந்தினை பெற்றுக்கொள்ள வேண்டும். இந்த ஆய்வு சம்பந்தமான ஆய்வக பரிசோதனைகள் கட்டணமின்றி செய்யப்படும். 40 நாட்கள் மருந்து உட்கொள்ளும் காலம் முடிந்த பிறகு நோய்க்கான குறிகுணங்கள் மற்றும் ஆய்வக பரிசோதனைகள் இவற்றின் முடிவுகளின் அடிப்படையில் மருந்தின் பரிகரிப்புத்திறன் கண்டறியப்படும்.

இந்த ஆய்வு சம்பந்தமாக சில கேள்விகளை தங்களிடம் கேட்க இருக்கிறேன். தங்களிடமிருந்து பெறப்படும் கருத்துக்கள் மற்றும் குறிப்புகள் அனைத்தும் நம்பிக்கையாக பதிவு செய்யப்படும். இந்த ஆய்வில் தங்களை உட்படுத்திக்கொள்வதின் மூலம் எந்த வகையிலும் பாதிப்புக்குள்ளாக மாட்டீர்கள் என உறுதி அளிக்கிறேன்.

எந்தவித வற்புறுத்தலுமின்றி, இந்த ஆய்வில் பங்கேற்கவும், இந்த ஆய்வு சம்பந்தமாக கேட்கப்படும் கேள்விகளுக்கு பதில் கூறவும் தங்களுக்கு முழு சுதந்திரம் அளிக்கப்படுகிறது. இந்த ஆய்வில் பங்கேற்பதற்கு எந்த சன்மானமும் வழங்கப்படமாட்டாது. ஆனால், ஆய்வு முழுவதும் எனது மேற்பார்வையிலும், தங்கள் உடல்நலன் குறித்த தனி கவனத்திலும் மேற்கொள்ளப்படும். மதுமேகம் நோய்க்கான புதிய மருந்தின் பரிகரிப்புத்திறனை சமூகத்திற்கு உணர்த்தும் வகையில் இந்த ஆய்வு மேற்கொள்ளப்படுகிறது. இந்த ஆய்வினைத் தொடர தங்களுக்கு விருப்பம் இல்லையெனில், எப்பொழுது வேண்டுமானலும் ஆய்வின் இடையில் விலகிக்கொள்ளவும், மருத்துவமனையில் வழங்கப்படும் இந்நோய்க்கான வழக்கமான மருந்துகளை பெற்றுக்கொள்ளவும் அறிவுறுத்தப்படுகிறீர்கள்.

இந்த ஆய்வில் சேகரிக்கப்படும் விபரங்கள் அனைத்தும் தங்களுக்கும் முதன்மை ஆராய்ச்சியாளரான எனக்கும் இடையில் இரகசியமாக வைக்கப்படும். கேள்வி பதில் வடிவத்தில் தங்களிடம் கேள்விகள் கேட்கப்படும். அனைத்துப் படிவங்களிலும் தங்களின் பெயர் தவிர்க்கப்பட்டு ஆய்வாளரால் தங்களுக்கென தனிக் குறியீடு வழங்கப்படும். அந்தக் குறியீடு ஆய்வாளருக்கு மட்டுமே தெரிந்ததாக இருக்கும். நீங்கள் இந்த ஆய்வில் பங்கேற்க விருப்பப்பட்டால், திட்டவரைவுபடி தேர்வு செய்யப்படுவீர்கள்.

நீங்கள் இந்த ஆய்வில் பங்கேற்கும் முன், இந்த ஆய்வினைப் பற்றிய மேலும் விபரங்கள் பெற வேண்டுமென விருப்பப்பட்டால், இந்த ஆய்வின் முதன்மை ஆராய்ச்சியாளர் மற்றும் தேசிய சித்த மருத்துவமனை, பட்ட மேற்படிப்புத்துறை மாணவர் Dr.ச.கோ.செந்தில் குமார் ஆகிய என்னை 9994172837 என்ற எண்ணில் தொடர்பு கொள்ளலாம். மேலும், நீங்கள் இந்த ஆய்வில், உங்களது பங்கேற்பு மற்றும் உரிமை பற்றி தெரிந்து கொள்ள தேசிய சித்த மருத்துவமனை, தலைவர்/செயற்குழு உறுப்பினர் அவர்களையும் 91-44-22411611 என்ற எண்ணில் தொடர்பு கொள்ளலாம்.

OBSERVATION OF CLINICAL LABORATORY EXAMINATIONS

At the time of admission to the trial, in all the 40 patients the following parameters observed,

Haematology

- Hb (gms%)
- Total WBC Count(cells/cumm)
- DC
  - Polymorphs(%)
  - Lymphocytes (%)
  - Eosinophils (%)
  - Monocytes (%)
  - Basophils (%)
- Total RBC count (cells/cu.mm)
- ESR(mm/hr)

Renal function test

- Blood urea (mg/dl)
- S. Creatinine (mg/dl)
- Uric acid (mg/dl)

Lipid profile

- S. Total cholesterol (mg/dl)
- HDL (mg/dl)
- LDL (mg/dl)
- VLDL (mg/dl)
- TGL (mg/dl)

Liver function test

- S. Total bilirubin (mg/dl)
- S. Direct bilirubin (mg/dl)
- S. Indirect bilirubin (mg/dl)
- SGOT (U/dl)
- SGPT (U/dl)
- S. Alkaline phosphatase (U/dl)
- S. Total protein (g/dl)
- S. Albumin (g/dl)
- S. Globulin (g/dl)

Other test

- S. Calcium (mg/dl)
- S. Phosphorous (mg/dl)

Urine examination

- Neerkuri & Neikuri
- Albumin
- Sugar (Fasting & postprandial)
- Deposits

Specific investigation

- OGTT
    - Fasting (over night fast)
    - 2 Hours after glucose load

Before treatment- Haematology

S.No	OP/IP No	Age/ Sex	B.Sugar mg/dl		Hb gms%	TC cu/mm	DC-%				ESR mm/hr		T.RBC million
			FA	PP			P	L	E	M	1/2	1	
1.	C79506	52/F	135	210	13.6	7400	61	34	03	-	6	12	3.1
2.	C93762	48/F	140	235	14.6	8300	55	40	05	-	10	20	3.4
3.	C29305	50/F	160	230	15.5	7900	55	40	03	-	08	16	3.2
4.	C98043	40/F	138	210	13.5	6600	62	34	04	-	2	4	5.2
5.	C28386	50/F	131	212	13.3	7200	67	28	05	-	4	8	4.2
6.	C41076	44/F	176	278	12.9	6300	49	45	06	-	4	12	4.2
7.	C61757	45/F	131	266	12	7300	56	39	05	-	08	16	3.1
8.	4244	45/F	126	219	13.3	10000	74	20	06	-	22	62	4.4
9.	4151	55/F	142	272	12.5	9500	54	41	05	-	28	60	3.9
10.	4182	53/F	156	252	11.6	7300	70	26	04	-	2	50	4.2
11.	4210	40/F	127	226	13.3	6900	62	36	02	-	08	15	4.2
12.	4203	55/F	127	210	10.0	7700	67	30	03	-	28	76	3.4
13.	4129	48/F	138	261	13.7	6700	60	37	03	-	10	26	4.7
14.	4236	55/F	134	300	11.3	10200	68	29	03	-	12	30	3.9
15.	C77100	50/M	136	239	14.9	4300	60	34	06	-	12	26	4.9
16.	C93323	40/M	136	254	14.5	6200	58	37	05	-	2	4	5.0
17.	C77872	33/M	117	257	14.8	8800	71	23	06	-	6	14	4.9
18.	C76014	43/M	157	245	14	5500	66	30	04	-	2	4	5.1
19.	C73821	43/M	124	200	16.8	7400	53	44	03	-	8	16	5.6
20.	C77322	53/M	134	236	15.5	7300	55	40	05	-	4	10	4.8

Before treatment- Haematology

S.No	OP/IP No	Age/ Sex	B.Sugar mg/dl		Hb gms%	TC cu/mm	DC-%				ESR mm/hr		T.RBC million
			FA	PP			P	L	E	M	1/2	1	
21	C84379	40/M	173	265	14.9	7700	58	41	01	-	4	8	5.0
22.	C75253	36/M	128	237	15.4	7800	53	39	08	-	4	8	5.5
23.	B95665	42/M	145	213	11.0	8000	66	32	04	-	04	08	4.8
24.	C92670	46/M	160	270	10.7	8200	52	44	03	01	20	40	3.5
25.	C99837	41/M	128	268	17.2	5900	69	25	05	-	2	4	5.6
26	4272	54/F	138	233	9.3	10100	73	22	05	-	6	20	3.5
27.	C81031	53/M	172	260	18	6400	48	45	07	-	2	4	5.8
28.	C92864	38/M	162	258	15	7300	68	27	05	-	4	20	5
29.	C42893	43/M	131	278	13.5	6100	50	44	06	-	2	6	4.4
30	C85416	42/M	160	220	15.3	5500	60	36	04	-	2	4	5.1
31.	C89234	50/M	159	298	16.1	7500	60	35	03	02	4	8	5.3
32.	4125	50/F	191	234	13.3	6500	56	39	05	-	2	4	4.5
33.	4035	52/F	169	239	14.3	8500	64	30	06	-	2	4	4.9
34.	B53440	42/F	168	295	13.6	10800	65	28	05	02	6	14	5.2
35.	C77041	51/M	132	262	14.4	8700	60	34	05	-	2	4	4.3
36.	C91330	53/M	148	300	14.5	6800	47	48	05	-	2	8	4.7
37.	C86489	55/M	146	178	16.7	10300	54	31	15	-	2	8	5.8
38.	B95283	50/M	132	300	15.9	10700	74	23	03	-	6	12	5.1
39.	C78746	42/M	160	290	14.6	7600	59	37	03	-	4	10	5.5
40.	C90731	45/M	180	295	7.5	8000	64	30	05	01	08	16	2.5

After treatment- Haematology

S.No	OP/IP No	Age/ Sex	B.Sugar mg/dl		Hb gms%	TC cu/mm	DC-%				ESR mm/hr		T.RBC million
			FA	PP			P	L	E	M	1/2	1	
1.	C79506	52/F	115	161	12.8	7200	58	32	01	-	05	11	3.0
2.	C93762	48/F	118	170	13.0	8200	54	43	03	-	07	13	3.2
3.	C29305	50/F	125	154	15.0	8300	56	42	03	-	05	12	3.1
4.	C98043	40/F	121	174	13.5	7600	63	38	02	-	09	18	3.3
5.	C28386	50/F	120	196	11.5	7400	64	32	04	-	6	14	4.2
6.	C41076	44/F	121	198	11.5	6300	40	55	05	-	4	8	4.2
7.	C61757	45/F	122	161	12.2	8500	53	45	02	-	8	26	4.4
8.	4244	45/F	115	170	12.4	8100	53	47	03	-	11	22	3.2
9.	4151	55/F	126	152	11.8	7300	52	41	01	-	03	06	3.7
10.	4182	53/F	112	167	13.7	8500	65	36	03	-	04	08	4.1
11.	4210	40/F	112	123	11.7	7800	66	32	02	-	02	06	4.4
12.	4203	55/F	122	136	9.2	9300	69	27	04	-	30	72	3.9
13.	4129	48/F	110	135	12.1	11500	65	30	05	-	2	4	4.7
14.	4236	55/F	107	211	9.8	7400	64	31	05	-	16	36	3.6
15.	C77100	50/M	126	146	13.4	4500	55	40	05	-	6	14	5.1
16.	C93323	40/M	112	164	11.8	7600	52	45	02	-	06	12	3.2
17.	C77872	33/M	110	190	12.7	11200	80	18	02	-	2	4	4.9
18.	C76014	43/M	120	170	15.1	6700	57	40	03	-	4	8	5.0
19.	C73821	43/M	118	177	15.5	7300	61	33	06	-	10	22	5.3
20.	C77322	53/M	121	177	15.9	7900	58	37	05	-	2	4	4.8

After treatment - Haematology

S.No	OP/IP No	Age/ Sex	B.Sugar mg/dl		Hb gms%	TC cu/mm	DC-%				ESR mm/hr		T.RBC million
			FA	PP			P	L	E	M	1/2	1	
21	C84379	40/M	123	248	15.0	8200	52	45	03	-	2	6	5.1
22.	C15253	36/M	116	207	14.9	8800	53	43	04	-	4	6	5.3
23.	B95665	42/M	122	164	12.6	9000	62	37	05	-	04	08	4.2
24.	C92670	46/M	126	177	10.2	8500	57	41	06	-	10	20	3.8
25.	C99837	41/M	112	187	15.1	5700	62	33	05	-	2	8	5.6
26	4272	54/F	130	192	11.6	7400	58	37	02	-	08	16	4.2
27.	C81031	53/M	140	211	16.4	5600	60	37	03	-	4	8	5.8
28.	C92864	38/M	139	198	13.9	8200	68	30	02	-	4	8	5.8
29.	C42893	43/M	130	196	14	4600	45	50	05	-	2	4	4.6
30	C85416	42/M	139	180	15.4	7100	65	29	06	-	4	8	5.2
31.	C89234	50/M	140	182	14.7	7100	59	35	06	-	4	8	4.9
32.	4125	50/F	185	336	14.8	9200	65	29	05	01	2	8	5.1
33.	4035	52/F	92	126	12.6	1800	52	44	03	-	06	12	4.1
34.	B53440	42/F	143	225	13.4	9400	64	34	02	-	8	30	5.1
35.	C77041	51/M	154	220	14	8100	57	37	05	-	5	10	4.5
36.	C91330	53/M	148	220	15	7400	46	50	04	-	8	18	4.7
37.	C86489	55/M	156	280	14.6	9900	66	20	14	-	2	4	5.6
38.	B95283	50/M	149	248	15.3	7600	61	36	03	-	2	6	4.9
39.	C78746	42/M	252	300	13.2	9200	61	32	04	01	08	16	4.2
40.	C90731	45/M	147	191	9.8	8200	53	30	04	-	06	12	3.5

Before treatment- Haematology

S. No	OP/IP No	Age/ Sex	Lipid profile					Liver Function Test												RFT	
			T.Cho Mg/dl	HDL Mg/dl	LDL Mg/dl	VLDL Mg/dl	TGL Mg/dl	T. Bmg/dl	D. Bmg/dl	I.B mg/dl	OT u/l	PT u/l	ALK u/l	T.PGms %	Alb Gms %	Glo Gms %	Cal Mg/ dl	P Mg/dl	U. A Mg/dl	U mg/dl	Cr Mg/dl
1.	C79506	52/F	244	45	111	12	60	0.6	0.2	0.4	36	24	236	8.6	4.4	4.2	8.2	3.5	5.0	26	0.6
2.	C93762	48/F	288	50	142	68	342	0.5	0.3	0.2	30	26	216	8.2	4.6	3.6	8.4	3.6	4.9	38	0.6
3.	C29305	50/F	218	45	112	29	148	0.6	0.2	0.4	34	27	195	7.9	4.8	3.1	8.2	3.4	4.7	18	0.8
4.	C98043	40/F	288	50	142	68	342	0.4	0.2	0.2	25	27	142	6.4	3.7	2.8	10	2.9	3.1	16	0.5
5.	C28386	50/F	110	26	68	28	140	0.5	0.2	0.3	29	30	162	7.0	3.0	4.0	10	2.9	4.4	20	0.6
6.	C41076	44/F	206	37	152	44	220	0.4	0.2	0.2	20	22	160	6.4	3.9	2.5	11	3.5	4.6	20	0.7
7.	C61757	45/F	188	31	106	33	165	0.6	0.3	0.3	34	48	235	8.6	4.8	3.8	10	3.0	5.2	18	0.6
8.	4244	45/F	169	33	77	32	163	0.7	0.3	0.4	12	14	155	6.6	3.7	2.9	10	2.8	4.0	15	0.4
9.	4151	55/F	197	35	115	50	251	0.5	0.2	0.3	17	19	166	5.0	2.0	3.0	10	2.9	3.7	17	0.5
10.	4182	53/F	179	36	92	46	234	0.7	0.3	0.4	16	18	163	6.5	4.0	2.5	11	3.1	3.4	20	0.6
11.	4210	40/F	171	41	112	44	224	0.4	0.2	0.2	33	36	201	7.0	4.0	3.0	10	2.9	5.5	26	0.7
12..	4203	55/F	218	45	112	29	148	0.5	0.2	0.3	13	15	176	6.7	3.8	2.9	10	2.7	5.1	27	0.8
13.	4129	48/F	227	42	107	45	229	0.5	0.2	0.3	20	22	172	6.5	3.2	3.3	10	3.0	3.5	15	0.5
14.	4236	55/F	246	39	106	32	17	0.8	0.4	0.4	16	18	172	5.5	3.5	2.0	8.3	4.0	3.7	17	0.5
15.	C77100	50/M	212	36	102	41	209	0.6	0.2	0.4	43	44	196	6.0	4.0	2.0	10	3.1	6.9	14	0.4
16.	C93323	40/M	117	31	70	38	193	0.5	0.2	0.3	12	16	151	5	3	2	10	3.2	3.2	19	0.5
17.	C77872	33/M	154	58	92	38	193	0.6	0.2	0.4	23	25	232	7.8	3.2	4.6	11	3.1	4.7	20	0.7
18.	C76014	43/M	230	45	115	63	257	0.6	0.3	0.3	22	18	160	7.2	5.2	2.0	10	3.2	3.4	27	1.2
19.	C73821	43/M	168	39	114	28	54	0.6	0.2	0.4	28	29	178	6.8	4.0	2.8	10	3.2	7.4	15	0.4
20.	C77322	53/M	228	41	123	64	320	0.7	0.3	0.4	38	33	190	8.6	4.6	4.0	9.2	3.8	4.0	22	0.6

Before treatment- Haematology

S. No	OP/IP No	Age/ Sex	Lipid profile					Liver Function Test												RFT	
			T.Cho Mg/dl	HDL Mg/dl	LDL Mg/dl	VLDL Mg/dl	TGL Mg/dl	T. Bmg/dl	DB mg/dl	I.B mg/dl	OT u/l	PT u/l	ALK u/l	T. PGms %	Alb Gms %	Glo Gms %	Cal Mg/dl	P Mg/dl	U.A Mg/dl	U mg/dl	Cr Mg/dl
21.	C84379	40/M	201	34	126	31	158	0.6	0.2	0.4	20	22	160	7.0	4.0	3.0	10	2.9	4.8	27	0.7
22.	C15253	36/M	198	35	150	43	218	0.6	0.2	0.4	30	32	204	6.0	4.0	2.0	10	3.0	3.6	23	0.7
23.	B95665	42/M	213	50	96	37	212	0.8	0.4	0.4	28	21	211	8.4	4.8	3.6	7.8	3.2	5.1	27	0.7
24.	C92670	46/M	189	40	146	60	219	0.6	0.3	0.3	26	19	270	7.4	4.3	3.1	9.2	3.6	4.5	26	0.6
25.	C99837	41/M	163	32	81	37	185	0.8	0.3	0.5	21	25	198	7.0	4.0	3.0	11	3.0	4.3	18	0.5
26.	4272	54/F	102	22	50	46	234	0.4	0.2	0.2	13	16	190	6.1	3.8	2.3	10	2.7	6.5	45	1.0
27.	C81031	53/M	156	33	79	26	131	0.7	0.3	0.4	16	19	166	6.0	4.0	2.0	10	2.9	3.0	14	0.4
28.	C92864	38/M	146	32	86	24	121	0.7	0.2	0.5	17	19	174	6.5	4.2	2.3	11	3.1	3.3	30	1.0
29.	C42893	43/M	188	34	86	78	393	0.4	0.2	0.2	13	15	165	6.7	4.9	1.8	11	3.6	3.3	25	0.7
30.	C85416	42/M	247	50	135	60	300	0.5	0.2	0.3	22	23	155	7.2	5.2	2.0	10	3.2	8.2	22	0.6
31.	C89234	50/M	195	40	108	52	263	0.6	0.2	0.4	21	22	220	6.6	4.3	2.3	11	3.0	7.5	31	0.8
32.	4125	50/F	172	35	48	45	225	0.5	0.2	0.3	22	26	188	7.3	4.3	3.0	10.	3.0	6.3	24	0.7
33.	4035	52/F	171	45	95	31	187	0.5	0.3	0.2	33	42	213	8.9	4.8	4.1	7.6	3.8	5.8	21	0.9
34.	B53440	42/F	243	50	142	51	257	0.5	0.2	0.3	50	78	212	6.9	4.2	2.7	8.7	3.1	6.7	14	0.4
35.	C77041	51/M	149	32	94	23	119	0.7	0.3	0.4	14	15	130	6.6	4.2	2.4	11	3.0	5.1	14	0.5
36.	C91330	53/M	185	36	91	62	311	1.0	0.5	0.5	22	28	204	7.2	5.2	2.0	11	3.7	4.0	27	0.7
37.	C86489	55/M	155	30	89	44	222	0.6	0.2	0.4	24	25	188	7.0	5.0	2.0	10	3.0	6.3	19	0.5
38.	B95283	50/M	244	45	111	12	60	0.7	0.3	0.4	11	12	156	6.8	4.0	2.8	10	2.9	5.3	32	0.9
39.	C78746	42/M	192	23	166	25	125	0.6	0.2	0.4	24	26	238	7.0	4.8	2.2	9.3	3.5	4.9	20	0.6
40.	C90731	45/M	264	41	164	56	227	0.5	0.3	0.2	16	18	212	7.6	4.3	3.3	8.9	3.2	3.1	22	0.7

After treatment - Haematology

S. No	OP/IP No	Age/ Sex	Lipid profile					Liver Function Test											RFT	
			T.Cho Mg/dl	HDL Mg/dl	LDL Mg/dl	VLDL Mg/dl	TGL Mg/dl	T. Bmg/dl	DB mg/dl	IB mg/dl	OT u/l	PT u/l	ALK u/l	T.PGms %	Alb Gms %	Glo Gms %	Cal Mg/dl	P Mg/dl	U. A Mg/dl	U mg/dl	Cr Mg/dl
1.	C79506	52/F	169	40	98	37	186	0.8	0.6	0.2	36	22	240	8.4	4.2	4.2	8.0	3.2	5.1	26	0.6
2.	C93762	48/F	203	47	118	26	126	0.5	0.2	0.3	34	24	215	8.6	4.6	3.8	8.6	3.4	5.0	30	0.9
3.	C29305	50/F	186	35	100	40	166	0.6	0.3	0.3	28	26	210	8.0	4.8	3.2	8.2	3.2	5.1	20	0.6
4.	C98043	40/F	115	41	74	37	213	0.5	0.3	0.2	34	30	215	8.4	5.0	3.4	8.0	3.6	4.2	26	0.8
5.	C28386	50/F	139	30	70	33	169	0.4	0.2	0.2	16	17	156	6.0	3.7	2.3	10	2.9	4.4	20	0.6
6.	C41076	44/F	203	47	118	26	129	0.4	0.2	0.2	13	16	149	6.1	3.9	2.2	10	3.0	3.0	18	0.6
7.	C61757	45/F	165	40	94	35	175	0.4	0.2	0.2	20	21	182	5.7	2.9	2.8	10	2.9	3.0	26	0.7
8.	4244	45/F	147	32	78	23	118	0.6	0.3	0.3	26	22	260	8.2	4.8	3.4	9.0	3.4	4.2	26	0.6
9.	4151	55/F	126	30	88	35	176	0.8	0.4	0.4	32	36	210	8.4	4.6	3.8	8.2	3.4	5.0	20	0.6
10.	4182	53/F	170	36	113	31	157	1.0	0.5	0.5	26	20	215	8.2	4.2	4.0	9.2	4.2	4.0	22	0.7
11.	4210	40/F	200	34	120	76	322	0.7	0.3	0.4	20	21	163	6.8	4.0	2.8	10	3.0	5.0	19	0.6
12..	4203	55/F	201	44	120	36	166	0.5	0.2	0.3	14	15	151	7.0	4.0	3.0	8.6	2.9	5.4	24	0.8
13.	4129	48/F	205	40	105	45	170	0.6	0.2	0.4	24	26	210	7.0	4.0	3.0	10	3.3	7.3	26	0.8
14.	4236	55/F	229	42	108	22	111	0.7	0.3	0.4	22	24	170	5.5	3.5	2.0	10	3.2	4.5	20	0.6
15.	C77100	50/M	179	35	86	36	179	0.6	0.2	0.4	24	26	179	6.0	4.0	2.0	10	3.1	4.1	14	0.4
16.	C93323	40/M	156	31	74	30	122	0.6	0.3	0.3	26	18	210	8.4	4.6	3.8	8.4	3.9	4.7	16	0.8
17.	C77872	33/M	162	33	88	31	158	0.6	0.2	0.4	11	12	141	6.3	4.0	2.3	10	3.0	3.5	24	0.7
18.	C76014	43/M	190	41	98	55	212	0.5	0.2	0.3	22	23	155	7.0	5.0	2.0	10	3.5	4.1	21	1.2
19.	C73821	43/M	153	30	74	12	61	0.6	0.2	0.4	22	24	226	6.4	4.0	2.4	10	3.0	6.0	15	0.5
20.	C77322	53/M	176	31	101	47	236	0.8	0.3	0.5	18	20	145	5.0	3.0	2.0	10	3.1	3.9	22	0.6

After treatment - Haematology

S. No	OP/IP No	Age/ Sex	Lipid profile					Liver Function Test												RFT	
			T.Cho Mg/dl	HDL Mg/dl	LDL Mg/dl	VLDL Mg/dl	TG mg/dl	TB mg/dl	DB mg/dl	I.B mg/dl	OT u/l	PT u/l	ALK u/l	T.PGms %	AlbGms %	Glo Gms %	Cal Mg/dl	P Mg/dl	U.A Mg/dl	U mg/dl	Cr Mg/dl
21.	C84379	40/M	185	38	92	27	138	0.7	0.3	0.4	20	22	160	7.0	4.0	3.0	10	2.9	4.8	27	0.7
22.	C15253	36/M	170	36	113	31	157	0.7	0.3	0.4	20	23	155	5.0	3.0	2.0	10	3.1	5.3	20	0.6
23.	B95665	42/M	115	41	61	30	164	0.6	0.4	0.2	28	21	215	8.4	4.2	4.2	7.6	3.4	5.4	30	0.5
24.	C92670	46/M	164	31	116	39	166	0.6	0.3	0.3	30	26	280	8.6	5.0	3.0	8.3	3.4	5.4	24	0.7
25.	C99837	41/M	147	32	78	23	118	0.6	0.2	0.4	20	22	228	5.4	3.4	2.0	10	3.0	4.5	16	0.4
26.	4272	54/F	154	30	74	37	129	0.8	0.4	0.4	30	34	250	8.2	4.2	4.0	8.5	3.0	5.1	36	0.6
27.	C81031	53/M	189	36	92	18	90	0.4	0.2	0.2	25	27	176	6.1	4.0	2.1	10	3.1	3.0	14	0.4
28.	C92864	38/M	192	36	100	16	81	0.9	0.3	0.6	20	22	187	5.1	3.0	2.1	10	3.0	5.1	20	0.6
29.	C42893	43/M	176	30	94	41	190	0.5	0.3	0.2	22	38	197	9.0	5.2	3.8	7.9	3.8	5.2	26	0.8
30.	C85416	42/M	180	40	96	37	170	0.6	0.2	0.4	20	21	160	6.4	4.2	2.2	10	3.1	4.1	18	0.5
31.	C89234	50/M	169	40	98	37	186	0.5	0.2	0.3	12	15	177	6.0	4.0	2.0	9.9	2.9	3.8	14	0.4
32.	4125	50/F	186	35	100	40	203	0.5	0.2	0.3	12	15	137	7.0	5.0	2.0	9.8	2.6	4.3	21	0.6
33.	4035	52/F	152	46	87	19	157	0.6	0.4	0.2	40	32	310	7.4	3.2	4.2	8.2	3.2	5.2	19	0.6
34.	B53440	42/F	193	40	98	55	279	0.6	0.2	0.4	28	30	315	7.5	4.3	3.2	8.5	3.1	4.8	19	0.7
35.	C77041	51/M	115	30	61	17	85	0.6	0.2	0.4	14	15	226	5.9	3.0	2.9	11	3.0	3.3	14	0.4
36.	C91330	53/M	155	31	75	37	185	0.4	0.2	0.2	12	14	166	7.5	4.8	2.7	11	3.1	3.6	22	0.6
37.	C86489	55/M	160	34	72	33	166	1.5	0.6	0.9	12	13	160	6.4	3.7	2.7	10	3.2	4.3	19	0.6
38.	B95283	50/M	189	33	100	24	121	0.5	0.2	0.3	20	22	167	5.8	3.0	2.8	9.8	2.7	2.9	20	0.6
39.	C78746	42/M	126	30	88	35	176	0.8	0.3	0.5	19	20	172	6.5	3.6	2.9	10	2.9	3.5	14	0.4
40.	C90731	45/M	197	30	111	43	116	0.6	0.3	0.3	26	22	222	7.4	4.6	3.4	9.0	4.6	3.6	18	0.7

Before treatment – Urine analysis

S.NO	OP/IP No	Age/ Sex	Alb	Sugar		Deposit	Neerkkuri	Neikkuri
				F	PP			
1.	C79506	52/F	Nil	N	++	1-2Pc,2-4Ec	Straw color	Slowly spread
2.	C93762	48/F	Nil	T	++	1-2Pc,2-4Ec	Straw color	Slowly spread
3.	C29305	50/F	Nil	T	++	2-3Pc,1-2Ec	Straw color	Slowly spread
4.	C98043	40/F	Nil	N	+	2-4Pc,2-6Ec	Straw color	Slowly spread
5.	C28386	50/F	Nil	N	+	2-4Pc,4-5Ec	Straw color	Slowly spread
6.	C41076	44/F	Nil	N	++	1-2Pc,2-4Ec	Straw color	Slowly spread
7.	C61757	45/F	Nil	N	N	1-2Pc,1-3Ec	Straw color	Slowly spread
8.	4244	45/F	Nil	N	++	4-5Pc,5-6Ec	Straw color	Slowly spread
9.	4151	55/F	Nil	N	N	3-5Pc,2-3Ec	Straw color	Slowly spread
10.	4182	53/F	Nil	N	+	8-9Pc,6-8Ec	Straw color	Slowly spread
11.	4210	40/F	Nil	N	N	2-4Pc,2-4Ec	Straw color	Fastly spread
12..	4203	55/F	Nil	N	N	4-5Pc,4-5Ec	Yellowish	Slowly spread
13.	4129	48/F	Nil	N	N	1-3Pc,2-4Ec	Straw color	Slowly spread
14.	4236	55/F	Nil	+	++	3-5Pc,2-3Ec	Straw color	Fastly spread
15.	C77100	50/M	Nil	N	+	2-4Pc,1-2Ec	Straw color	Slowly spread
16.	C93323	40/M	Nil	N	+	2-3Pc,1-5Ec	Straw color	Slowly spread
17.	C77872	33/M	Nil	T	+	1-2Pc,2-4Ec	Straw color	Slowly spread
18.	C76014	43/M	Nil	N	+	2-3Pc,1-4Ec	Straw color	Slowly spread
19.	C73821	43/M	Nil	N	N	1-2Pc,2-3Ec	Straw color	Slowly spread
20.	C77322	53/M	Nil	N	++	4-5Pc,2-4Ec	Straw color	Slowly spread

Before treatment – Urine analysis

S.NO	OP/IP No	Age/ Sex	Alb	Sugar		Deposit	Neerkuri	Neikkuri
				F	PP			
21.	C84379	40/M	Nil	T	++	1-4Pc,2-5Ec	Straw color	Slowly spread
22.	C15253	36/M	Nil	N	T	2-3Pc,4-5Ec	Straw color	Slowly spread
23.	B95665	42/M	Nil	N	+	4-6Pc,2-4Ec	Straw color	Fastly spread
24.	C92670	46/M	Nil	+	++	2-4Pc,4-5Ec	Straw color	Slowly spread
25.	C99837	41/M	Nil	N	+	1-5Pc,2-3Ec	Straw color	Slowly spread
26.	4272	54/F	Nil	+	++	3-6Pc,3-6Ec	Straw color	Slowly spread
27.	C81031	53/M	Nil	T	+	2-3Pc,2-3Ec	Straw color	Slowly spread
28.	C92864	38/M	Nil	N	+	1-2Pc,2-3Ec	Straw color	Slowly spread
29.	C42893	43/M	Nil	N	++	2-4Pc,3-5Ec	Straw color	Fastly spread
30.	C85416	42/M	Nil	N	+	1-2Pc,2-8Ec	Straw color	Slowly spread
31.	C89234	50/M	Nil	T	+	2-3Pc,1-4Ec	Straw color	Fastly spread
32.	4125	50/F	Nil	+	++	3-4Pc,1-2Ec	Straw color	Slowly spread
33.	4035	52/F	Nil	N	++	5-6Pc,4-5Ec	Straw color	Slowly spread
34.	B53440	42/F	Nil	N	++	2-4Pc,1-2Ec	Straw color	Slowly spread
35.	C77041	51/M	Nil	N	++	2-5Pc,4-5Ec	Straw color	Slowly spread
36.	C91330	53/M	Nil	N	+	4-5Pc,4-5Ec	Yellowish	Slowly spread
37.	C86489	55/M	Nil	N	+	1-2Pc,2-6Ec	Straw color	Slowly spread
38.	B95283	50/M	Nil	N	+	2-3Pc,1-2Ec	Straw color	Slowly spread
39.	C78746	42/M	Nil	N	++	2-4Pc,8-9Ec	Straw color	Slowly spread
40.	C90731	45/M	Nil	+	++	1-2Pc,2-4Ec	Straw color	Slowly spread

Afetr treatment – Urine analysis

S.NO	OP/IP No	Age/ Sex	Alb	Sugar		Deposit	Neerkuri	Neikkuri
				F	PP			
1.	C79506	52/F	Nil	N	N	1-2Pc,1-2Ec	Straw color	Slowly spread
2.	C93762	48/F	Nil	N	+	1-2Pc,1-2Ec	Straw color	Slowly spread
3.	C29305	50/F	Nil	N	N	2-3Pc,2-4Ec	Straw color	Slowly spread
4.	C98043	40/F	Nil	N	N	1-2Pc,2-5Ec	Straw color	Slowly spread
5.	C28386	50/F	Nil	N	+	2-5Pc,4-5Ec	Straw color	Slowly spread
6.	C41076	44/F	Nil	N	+	1-3Pc,2-4Ec	Straw color	Slowly spread
7.	C61757	45/F	Nil	N	N	3-4Pc,1-3Ec	Straw color	Slowly spread
8.	4244	45/F	Nil	N	N	1-2Pc,2-4Ec	Straw color	Slowly spread
9.	4151	55/F	Nil	N	N	3-4Pc,2-5Ec	Straw color	Slowly spread
10.	4182	53/F	Nil	N	N	2-5Pc,2-3Ec	Straw color	Slowly spread
11.	4210	40/F	Nil	N	N	1-2Pc,2-5Ec	Straw color	Fastly spread
12..	4203	55/F	Nil	N	N	2-4Pc,2-5Ec	Straw color	Slowly spread
13.	4129	48/F	Nil	N	N	1-3Pc,2-4Ec	Straw color	Slowly spread
14.	4236	55/F	Nil	N	N	1-2Pc,2-4Ec	Straw color	Fastly spread
15.	C77100	50/M	Nil	N	N	2-3Pc,2-3Ec	Straw color	Slowly spread
16.	C93323	40/M	Nil	N	N	4-5Pc,1-4Ec	Straw color	Slowly spread
17.	C77872	33/M	Nil	N	+	4-5Pc,2-6Ec	Straw color	Slowly spread
18.	C76014	43/M	Nil	N	T	1-4Pc,1-2Ec	Straw color	Slowly spread
19.	C73821	43/M	Nil	N	N	1-2Pc,2-3Ec	Straw color	Slowly spread
20.	C77322	53/M	Nil	N	N	4-5Pc,1-5Ec	Straw color	Slowly spread

Afetr treatment – Urine analysis

S.NO	OP/IP No	Age/ Sex	Alb	Sugar		Deposit	Neerkuri	Neikkuri
				F	PP			
21.	C84379	40/M	Nil	N	+	1-4Pc,2-3Ec	Straw color	Slowly spread
22.	C15253	36/M	Nil	N	+	2-4Pc,1-4Ec	Straw color	Slowly spread
23.	B95665	42/M	Nil	N	N	3-5Pc,2-4Ec	Straw color	Slowly spread
24.	C92670	46/M	Nil	N	N	2-4Pc,4-6Ec	Straw color	Slowly spread
25.	C99837	41/M	Nil	N	+	4-6Pc,4-8Ec	Straw color	Slowly spread
26.	4272	54/F	Nil	N	+	2-8Pc,4-6Ec	Straw color	Slowly spread
27.	C81031	53/M	Nil	N	+	1-2Pc,1-2Ec	Straw color	Slowly spread
28.	C92864	38/M	Nil	N	+	4-5Pc,4-5Ec	Straw color	Slowly spread
29.	C42893	43/M	Nil	N	+	2-5Pc,2-3Ec	Straw color	Slowly spread
30.	C85416	42/M	Nil	N	T	1-2Pc,2-4Ec	Straw color	Slowly spread
31.	C89234	50/M	Nil	N	T	1-4Pc,4-5Ec	Straw color	Fastly spread
32.	4125	50/F	Nil	N	+	2-4Pc,1-2Ec	Straw color	Slowly spread
33.	4035	52/F	Nil	N	++	5-6Pc,6-9Ec	Straw color	Slowly spread
34.	B53440	42/F	Nil	N	++	3-5Pc,1-2Ec	Straw color	Fastly spread
35.	C77041	51/M	Nil	N	++	2-5Pc,1-2Ec	Straw color	Slowly spread
36.	C91330	53/M	Nil	N	++	2-4Pc,4-5Ec	Straw color	Slowly spread
37.	C86489	55/M	Nil	N	++	1-4Pc,2-6Ec	Straw color	Slowly spread
38.	B95283	50/M	Nil	N	++	2-5Pc,1-2Ec	Straw color	Slowly spread
39.	C78746	42/M	Nil	+	++	1-2Pc,1-2Ec	Straw color	Slowly spread
40.	C90731	45/M	Nil	T	++	2-4Pc,2-4Ec	Straw color	Slowly spread

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 600047
CERTIFICATE OF BOTANICAL AUTHENTICITY

Certified that the following plant drugs used in the Siddha formulation **Atthippattaiyathi Kasayam** (Internal) for the treatment of **Mathumegam** (Type – 2 Diabetes Mellitus) taken up for Post Graduation Dissertation studies by **Dr.S.G.Senthil Kumar**, M.D.(S), II year Department of Maruthuvam, 2011-12, are identified and authenticated through Visual inspection / Experience, Education & Training/ Organoleptic characters/ Morphology / Micromorphology / Taxonomical/ Microscopical methods.

Ficus racemosa Linn. (Moraceae), Bark

Cassia fistula Linn. (Caesalpiniaceae), Bark

Cassia auriculata Linn. (Caesalpiniaceae), Bark

Salacia oblonga Wall. (Celastraceae), Root bark

Madhuca longifolia(Linn.) Macbride. (Sapotaceae), Bark

Tamarindus indica Linn. (Caesalpiniaceae), Bark

Terminalia arjuna W. & A. (Combretaceae), Bark

Spermacose hispida Linn. (Rubiaceae), Root

Citrus medica Linn. (Rutaceae), Root

Hemidesmus indicus Schult. (Periplocaceae), Root

Amaranthus tricolor Linn. (Amaranthaceae), Root

Phyllanthus reticulatus Poir. (Euphorbiaceae), Root

Aloe barbadensis Mill. (Liliaceae), Root

Cyperus rotundus Linn. (Cyperaceae), Root tuber

Tinospora cordifolia (Willd.) Miers. (Menispermaceae), Stem

Zingiber officinale Rosc. (Zingiberaceae), Rhizome

Piper nigrum Linn. (Piperaceae), Fruit

Piper longum Linn. (Piperaceae), Fruit

Taxus baccata Linn. (Taxaceae), Leaf

Myristica fragrans Houtt. (Myristicaceae), Seed and Aril

Syzygium aromaticum(Linn.) Merr. & L.M. Perry (Myrtaceae), dried flower bud

Ferula foetida Regel. (Apiaceae), Gum-oleoresin

Certificate No: NIS/MB/44/2012

Date: 24-8-12

Authorized Signatory

Dr. D. ARAVIND, M.D.(s),M.Sc.,
Assistant Professor
Department of Medicinal Botany
National Institute of Siddha
Chennai - 600 047, INDIA

IAEC PROTOCOL NO: 1248/ac/09/CPCSEA/4-07/2011

20/12/2011

CERTIFICATE

This is certify that the project title Preclinical and clinical study on "Mathumegam" (Type-2 Diabetes Mellitus) and the drug of choice is "Aathipattaiyathi kasayam". **has been approved by the IAEC.**

Prof. Dr. K. Manickavasakam — **Name of Chairman/Member Secretary IAEC:**

Dr. B. Jayachandran Dare — **Name of CPCSEA nominee:**

Signature with date

Chairman/Member Secretary of IAEC:

CPCSEA nominee:

(Kindly make sure that minutes of the meeting duly signed by all the participants are maintained by Office)

NATIONAL INSTITUTE OF SIDDHA

(An Autonomous Body under Department of AYUSH)
Ministry Of Health & Family Welfare, Government of India

Tambaram Sanatorium, Chennai - 600 047
Tel : 044-22411611 Fax : 044-22381314
E-mail : nischennaisiddha@yahoo.co.in
Website : www.nischennai.org

Name: Dr.S.G.SENTHIL KUMAR., REG.NO: 32101207.
Title: Preclinical and Clinical study on MATHUMEGAM (DIABETES MELLITUS TYPE-2) and the drug of choice is ATTHIPPATTAIYATHI KASAYAM.

No. NIS/IEC/2011/3/07 - 24/12/2011

DECSION

Opinion of the Institutional Ethics Committee – Please Check one
✓ Approval _____ Modifications required prior to approval (Please specify one space below) _____ Disapproval Date of review: _____ Signed: _____ (Please print name) Dr.V.SUBRAMANIAN Chair person (Please delee as appropriate, Chairperson, Secretary)
Modifications needed Modification given to candidate
The research proponent is hereby informed that the Institutional Ethics Committee will require the following: 1. All adverse drug reactions (ADRs) that are both serious and unexpected to be reported promptly to the IEC within 7 working days 2. The progress report to be submitted to the IEC atleast annually 3. Upon completion of the study, a final study status report needs to submitted to the IEC

K. Manimekalai
(Dr. K. MANICKAVASAKAM)
Member secretary

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to ~~Mr/Ms~~/Dr.....S.G. SENTHIL KUMAR.....

for participating as a ~~Resource Person~~ / Delegate in the VII Workshop

on **"Research Methodology & Biostatistics"**

for AYUSH Post-Graduates & Researchers

organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University

from 6th Feb. 2012 to 10th Feb. 2012.

DR. MAYILVAHANAN NATARAJAN
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. (Orth. Onco.) F.R.C.S. (Eng) D.Sc.
7th VICE CHANCELLOR

Dr. R. SRILAKSHMI, DCH, Ph.D.
REGISTRAR

Dr. N. KABILAN, M.D. (Siddha)
READER, DEPT. OF SIDDHA